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Emission of Sulfur-Bearing Compounds from Motor Vehicle and Aircraft Engines. A Report to Congress

Biospherics, Inc, Rockville, MD

Prepared for

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Emission of Sulfur-Bearing Compounds from Motor Vehicle and Aircraft Engines

A Report to Congress

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A Report to Congress

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PREFACE

This report was written in accordance with Section 403(g) of the Clean Air Act as amended August 1977. Its purpose is to review the effects of sulfur-bearing compounds emitted from mobile sources and to analyze the costs and benefits of alternatives to control such emissions.

The report does not constitute an indepth scientific review of the published literature; rather, it presents an evaluation of the scientific data on the effects of the various sulfur compounds emitted by mobile sources. Mobile source emission rates are weighed against emission rates from stationary sources to determine the relative impact of these compounds on the public health and welfare.

The Agency is pleased to acknowledge the efforts of all persons and groups who have participated in preparing this document. In the last analysis, however, the Environmental Protection Agency is responsible for its content.

ABSTRACT

This report was generated in response to § 403(g) of The Clean Air Act as amended August, 1977. The report covers (1) a review of emission factors for sulfuric acid, sulfur dioxide, sulfate, hydrogen sulfide, and carbonyl sulfide from motor vehicles, motor vehicle engines and aircraft engines; (2) a review of the known effects on health and welfare of these compounds; (3) the status of technology to control such emissions; and (4) an analysis of the costs of control weighed against the social benefits of such control. Available emission factors for these pollutants were converted to ambient air concentrations by using dispersion and stochastic models. The predicted ambient air concentrations were compared to concentrations of these pollutants known to cause adverse health or welfare effects. Results of this comparison suggest that benefits of any control are likely to be small. Except for 3-way catalytic control technology, cost data for fuel desulfurization and vehicle on-board control technology suggest an extremely large economic impact. Consequently, specific controls of sulfur-bearing compounds from mobile sources are not recommended.

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1. INTRODUCTION

Sulfur is one of the most pervasive pollutants in the environment. In the United States alone, nearly 30 million tons of sulfur oxides are emitted annually into the atmosphere. At present, only about 2 percent of these emissions come from motor vehicles, but there is increasing concern that these mobile sources may exert significant effects on the public health and welfare. Not only are the types of sulfur compounds emitted by today's automobiles more toxic, but the quantity of sulfur emitted is likely to increase markedly over the next decade.

Shortly after the automotive industry chose the oxidation catalyst to control gaseous hydrocarbon and carbon monoxide emissions from automobiles, scientists from industry and government discovered that this device could convert exhaust sulfur dioxide into the more toxic compound sulfuric acid. This finding prompted the U.S. Environmental Protection Agency in 1974 to establish an interdisciplinary program to assess the environmental impact of emissions from vehicles equipped with oxidation catalytic control devices. A major portion of the Catalyst Research Program investigated the possibility that sulfuric acid and other exhaust products could accumulate on roadways and adversely affect the health of commuters.

A second concern involves the projected increase in production of diesel-powered vehicles, which are expected to comprise 25 percent of the new-car market by 1985. Diesel engines burn high-sulfur fuels, thereby emitting large amounts of sulfur dioxide and sulfuric acid into the environment.

The findings of the Catalyst Research Program and of independent investigations on health effects from sulfur-bearing compounds have been compiled in this special report to the United States Congress, under Section 403(g) of the 1977 Amendments to the Clean Air Act. Specifically, this report presents information on the emission rates of sulfur-bearing compounds from motor vehicles, motor vehicle engines, and aircraft engines, the potential for high ambient concentrations of these pollutants, the effects on health and welfare attributable to these concentrations, and an analysis of the costs and benefits of control.

2. EMISSIONS FACTORS

2.1 INTRODUCTION

Nationwide, the emissions of sulfur-related compounds, mostly in the form of sulfur oxides (SO_x), have shown a slight decline, from an estimated 29.1 million metric tons/year in 1970 to an estimated 26.9 million metric tons/year in 1976 (Hunt 1977). SO_x emissions from both transportation sources and stationary fuel combustion sources have remained almost constant over the period 1970-76, with transportation sources averaging 0.8 million metric tons/year, stationary sources averaging 21.8 million metric tons/year, and industrial processes averaging 5.3 million metric tons/year. The 1970-76 nationwide SO_x emission estimates are presented in Table 2-1. These estimates indicate that highway vehicles, hereinafter referred to as mobile sources, account for less than 2 percent of the total manmade atmospheric sulfur oxides. Although mobile source emissions to the atmosphere are small compared with those from stationary sources, it is possible though not probable that localized heavy concentrations of vehicular sulfur oxides could occur along portions of heavily traveled freeways, thereby resulting in a significant health hazard.

An estimate of ambient air exposure to emissions from mobile sources must consider variables of time, place, and degree of such exposure, as well as the nature of the vehicles and fuels studied. The localized atmospheric accumulation of automotive exhaust products is most likely to occur when three extreme conditions are met simultaneously:

Table 2-1. NATIONWIDE SULFUR OXIDE (SOx) EMISSION ESTIMATES, 1970-76
(millions of metric tons)

Source category	1970	1971	1972	1973	1974	1975	1976
Transportation	0.7	0.7	0.7	0.8	0.8	0.8	0.8
Highway vehicles	0.3	0.3	0.3	0.4	0.4	0.4	0.4
Nonhighway vehicles	0.4	0.4	0.4	0.4	0.4	0.4	0.4
Stationary fuel combustion	22.3	21.5	21.8	22.9	21.9	20.6	21.9
Electric utilities	15.7	15.6	16.0	17.5	17.0	16.7	17.6
Industrial	4.6	4.0	4.0	3.7	3.3	2.5	2.6
Residential, commercial, and institutional	2.0	1.9	1.8	1.7	1.6	1.4	1.7
Industrial processes	5.9	5.5	6.1	5.8	5.3	4.2	4.1
Chemicals	0.5	0.5	0.6	0.5	0.4	0.3	0.3
Petroleum refining	0.6	0.6	0.7	0.8	0.8	0.7	0.7
Metals	4.1	3.6	4.0	3.7	3.3	2.5	2.4
Mineral products	0.5	0.6	0.6	0.6	0.6	0.5	0.5
Oil and gas production and marketing	0.1	0.1	0.1	0.1	0.1	0.1	0.1
Other processes	0.1	0.1	0.1	0.1	0.1	0.1	0.1
Solid waste	0.1	0.1	0.1	0.1	0.1	0.0	0.0
Miscellaneous	0.1	0.1	0.1	0.1	0.1	0.1	0.1
Coal refuse burning	0.1	0.1	0.1	0.1	0.1	0.1	0.1
Total	29.1	27.9	28.8	29.7	28.2	25.7	26.9

Note: A zero indicates emissions of less than 50,000 metric tons per year.

Sources: National Air Quality and Emission Trends Report, 1976.
EPA Publication No. EPA-450/1-76-002, December 1977.

1. Atmospheric dispersion is limited by adverse weather conditions.
2. Dense traffic occurs.
3. Emission rates of particular pollutants are high.

Identification of the times and places in which vehicular pollutants are likely to be concentrated has centered around analysis of these factors. While dispersion conditions are mostly independent of roadway factors, emission rates are highly influenced by driving patterns, which in turn depend on traffic densit Therefore, it is important to measure emission factors over a variety of traffic situations or driving cycles, particularly those characteristic of high traffic density. Analysis of driving patterns characteristic of rush-hour traffic in southern California has resulted in a traffic model useful for estimating such emissions, the Crowded Urban Expressway (CUE) cycle.

The almost universal method to estimate overall traffic emissions involves measuring the mass of a pollutant emitted per vehicle mile traveled by a large number of cars. From individual vehicle measurements, an overall fleet emissions rate can be calculated to represent either an actual existing fleet (in the case of in-use vehicles) or a potential future fleet (in the case of prototype vehicles

From the fleet emissions factors, the traffic density, and the average vehicle speed, mass emissions from the overall roadway can be calculated for use in dispersion calculations. Therefore, the data collected here serve as overall fleet emissions factors for both real and projected future vehicle fleets.

The formation of sulfur-related exhaust emissions from mobile sources is a function of several variables, including the type of engine, the emissions control system, and the driving cycle. These variables notwithstanding, the fuel sulfur content is the critical factor. A number of studies have shown a linear relationship between the amount of sulfur in fuel and the amount of sulfur oxides emitted from the tailpipe (Krause et al. 1976; Braddock 1977; Braddock and Gabele 1977).

The mobile sources discussed in this report include gasoline-powered passenger cars, diesel-powered trucks and cars, and turbine aircraft. Data on the types and amounts of sulfur compounds emitted by these sources are summarized in Table 2-2. Data on the fuels used by them, including typical sulfur content, are presented in Table 2-3, along with the sulfur compounds monitored in exhaust.

2.2 EXPERIMENTAL METHODS

2.2.1 Dynamometer Test Procedures

Most vehicular exhaust emission studies have used chassis dynamometer techniques to simulate driving conditions based on the Federal Test Procedure (FTP), the Highway Fuel Economy Test (HWFET) procedure, and the Crowded Urban Expressway (CUE) procedure.

The FTP or city test simulates a 7.5-mile, stop-and-go trip with a speed range of 0 to 56 mph and an average speed of 20 mph. The trip takes 23 min and has 18 stops, with 19 percent of the trip spent idling, such as would be associated with city traffic lights or rush-hour driving. Two types of engine starts are

Table 2-2. SULFUR-BEARING EMISSIONS FROM MOBILE SOURCES

Mobile source type	$SO_4^=$ conversion rate, %	$SO_4^=$, mg/mile	SO_2, mg/mile	H_2S,[a] mg/mile	COS,[a] mg/mile	Comments
Non-catalyst cars	~1	~1	~100	Trace	≦0.6	All are CUE (Crowded Urban Expressway) emission rates normalized to 0.03 wt% sulfur fuel.[c]
Oxidation catalyst cars	~10	10-15	~65	≦7 ppm[b]	≦7 ppm[b]	
3-Way catalyst cars	~5	~4	~55	≦8	≦2.1	
Light-duty diesel cars	~2	~9	450	NT[d]	≦0.6	Using national average diesel fuel. Fuel sulfur level 0.23 wt%.
Heavy-duty diesel trucks	~2	~50	1500	NT	NT	
Aircraft gas turbine	~0.03	NA[e]	NA	NT	NT	Using 0.26 wt% sulfur level jet fuel.

[a] Values for H_2S and COS for catalysts in failure modes.
[b] Measured at idle.
[c] Estimated nationwide average for unleaded gasoline (Somers et al. 1977).
[d] NT = Not tested.
[e] NA = Not applicable.

NOTE: CONVERSION TO METRIC. To convert data expressed as grams per mile or milligrams per mile to grams or milligrams per kilometer, multiply the number given by 1.61.

Table 2-3. MOBILE SOURCES AND AVERAGE FUEL SULFUR LEVELS

Source type	Fuel type	Average fuel sulfur level in wt% sulfur	Reference[a]	Measured sulfur-bearing exhaust emission compounds
Non-catalyst vehicle	Regular leaded gasoline	0.038	1	SO_2, H_2SO_4, H_2S, COS
	Premium leaded gasoline	0.033	2	SO_2, H_2SO_4, H_2S, COS
Catalyst-equipped vehicle	Regular no-lead gasoline	0.024	1	SO_2, H_2SO_4, H_2S, COS
	Premium no-lead gasoline	0.014	1	SO_2, H_2SO_4, HS, COS
Diesel-powered vehicle	No. 2 diesel fuel	0.230	3	SO_2, H_2SO_4, COS
Aircraft gas turbine	Jet A-1 fuel	0.060	4	SO_2, H_2SO_4

[a] References: (1) Motor Vehicles Manufacturers Association, National Fuels Survey, 1977. (2) Motor Vehicles Manufacturers Association, National Fuels Survey, 1976. (3) E. M. Shelton, Diesel Fuel Oils, 1976, Petroleum Products Survey, ERDA Publication No. BERC/PPS-76/5, 1976. U.S. Energy Research and Development Administration, Bartlesville Energy Research Center. (4) E. M. Shelton, Aviation Turbine Fuels, 1976, Petroleum Products Survey, ERDA Publication No. BERC/PPS-77/2, April 1977. U.S. Energy Research Development Administration, Bartlesville Energy Research Center.

used in the FTP. The first simulates starting a car in the morning after it has been parked all night at an ambient temperature of 20°C. The other is a hot start, which simulates starting a vehicle after having parked it for 10 min. The results of the FTP are intended to represent the fuel economy under typical urban driving conditions.

The HWFET or highway test simulates a 10.25-mile, nonstop trip that begins with the vehicle warmed up. The trip has an average speed of 48 mph and lasts 13 min. Vehicle speed during the test ranges from 0 to 60 mph.

The CUE was designed to represent EPA's concept of conditions most conducive to high sulfate emissions, such as driving on a congested multilane freeway. The CUE simulates a 13.5-mile, stop-and-go driving cycle that begins with the vehicle warmed up. The schedule lasts 23 min, during which time speeds range from 0 to 57 mph and average 35 mph. The schedule contains 610, 567, and 221 sec of acceleration, deceleration, and cruise modes, respectively.

A comparison of the FTP, HWFET, and CUE test procedures is presented in Table 2-4, and speed-versus-time traces are depicted in Figure 2-1.

In the aircraft gas turbine emission test described in Section 2.3.3, the proposed 1979 Federal certification cycle was used (Fed. Reg. 38(136):19088, 1973). This cycle employs four simulated operating modes: idle, approach, climb, and takeoff.

*All data on emission rates in this report are expressed per mile. Multiply numbers by 1.61 to obtain a per kilometer number.

Table 2-4. COMPARISON OF DRIVING CYCLE CHARACTERISTICS

Driving cycle	Length, miles	Average speed, mph	Maximum speed, mph	Time, sec	No. of stops/cycle	Time at idle, %
FTP	7.50	20	57	1371	18	19.1
HWFET	10.25	48	60	765	2	1.0
CUE	13.50	35	57	1398	3	2.3

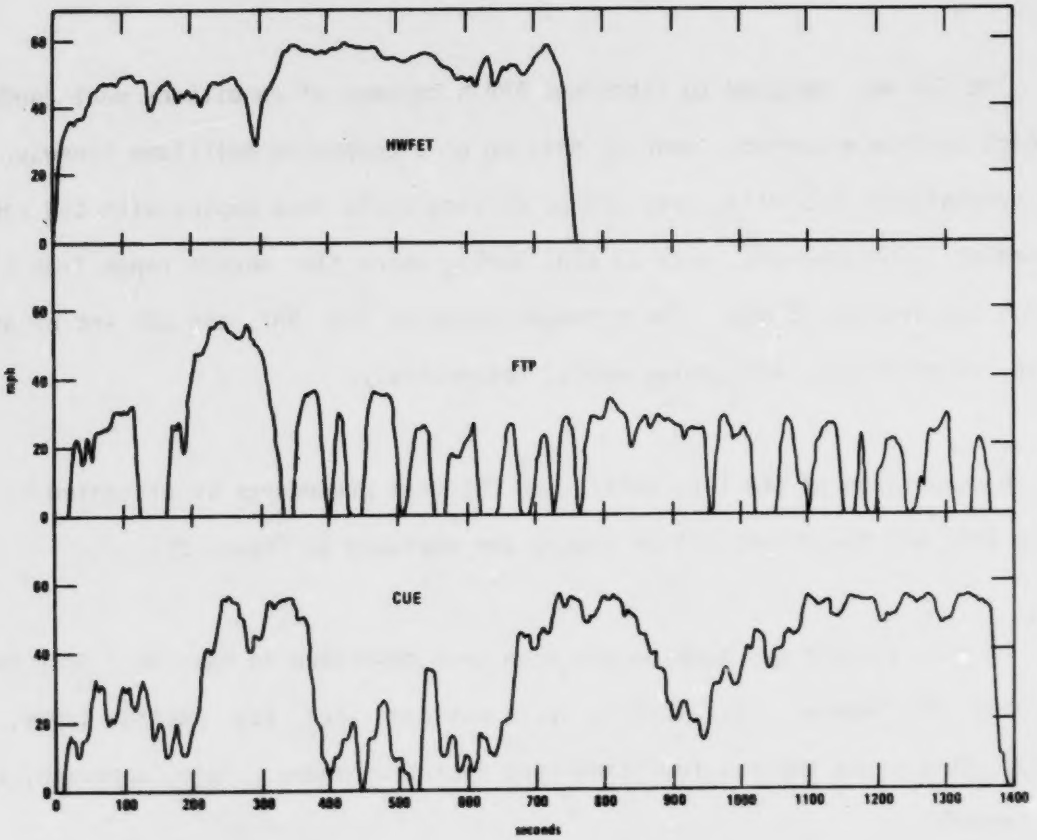

Figure 2-1. Speed-versus-time traces of FTP, HWFET, and CUE driving cycles.

2.2.2 Analytical Methods

Emissions estimation involves use of procedures to simulate performance of a mobile source, to sample exhaust, and to chemically analyze pollutant concentrations in samples.

Sampling of both gaseous and particulate sulfur components from motor vehicles, including SO_2, H_2SO_4, H_2S, and COS, has commonly been accomplished by utilizing the air dilution technique pioneered by Habibi. Use of this technique to measure sulfur compound emissions has been described by Bradow and Moran (1975) for catalyst-equipped cars, by Braddock and Bradow (1975) for diesel passenger cars, and by Hare et al. (1976) for diesel trucks and buses.

Figure 2-2 depicts a typical sampling system designed for the characterization and measurement of gaseous, particulate, and sulfur-related emissions from motor vehicles. The technique involves simulation of the air dilution which occurs as automobile exhaust enters the ambient air. Dilution and cooling causes low vapor pressure components such as H_2SO_4 to condense, and these condensates can then be trapped on filters for subsequent physical and chemical analysis.

Table 2-5 summarizes the various commonly used methods to analyze SO_2, H_2SO_4, H_2S, and COS in automobile exhausts.

In the aircraft gas turbine test described in Section 2.3.3, particulate emissions were extracted from the exhaust stream with a high-volume linear sampling rake and mixing plenum, and a stainless-steel line delivered emissions

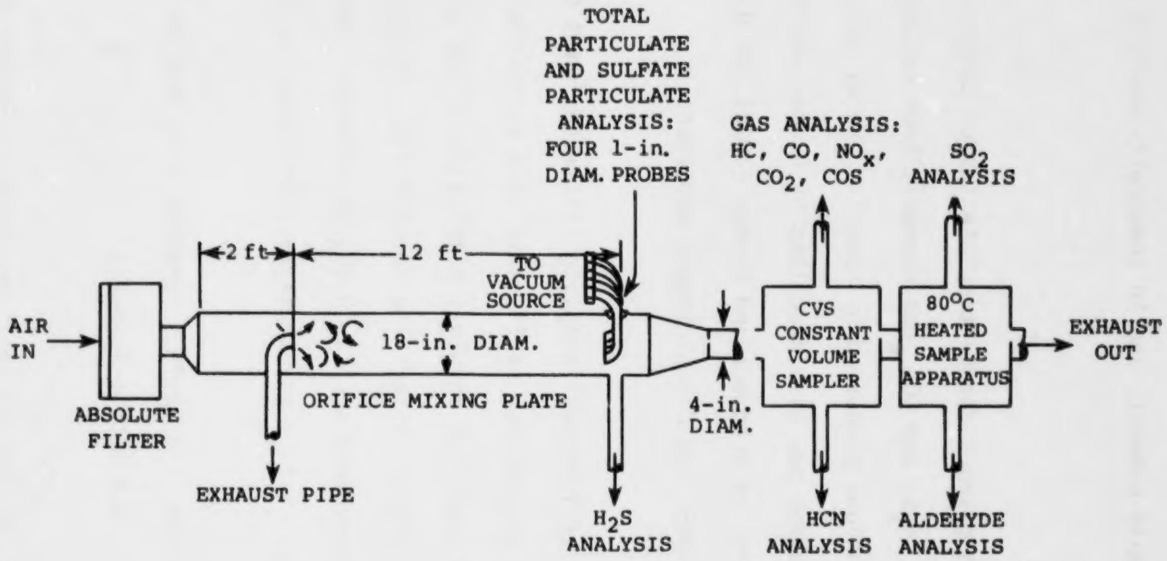
TOTAL PARTICULATE AND SULFATE PARTICULATE ANALYSIS: FOUR 1-in. DIAM. PROBES
GAS ANALYSIS: HC, CO, NO_x, CO_2, COS
SO_2 ANALYSIS
2 ft
12 ft
TO VACUUM SOURCE
AIR IN
18-in. DIAM.
CVS CONSTANT VOLUME SAMPLER
80°C HEATED SAMPLE APPARATUS
EXHAUST OUT
ABSOLUTE FILTER
ORIFICE MIXING PLATE
4-in. DIAM.
EXHAUST PIPE
H_2S ANALYSIS
HCN ANALYSIS
ALDEHYDE ANALYSIS

Figure 2-2. EPA-RTP dilution tunnel sampling system.

Table 2-5. SUMMARY OF THE COMMONLY USED METHODS FOR ANALYSIS OF SO_2, H_2SO_4, H_2S, AND COS

Sulfur compound	Type of method (type of detection)	Type of sample collected	Detection limit
SO_2[a]	Wet chemical (spectrophotometric)	Diluted exhaust (gaseous)	0.01 ppm
H_2SO_4[b]	Wet chemical (spectrophotometric)	47-mm filters (aerosol)	5 ng
H_2S[c]	Wet chemical (spectrophotometric or gas chromatographic)	Diluted or raw exhaust (gaseous)	0.01 ppm
COS[d]	Gas chromatographic	Diluted or raw exhaust (gaseous)	0.01 ppm

Sources: [a] Fed. Reg. 36(84):8187, 1971; Fed. Reg. 36(247):21893, 1971; Mulik et al. (1978).
[b] Tejada (1974); Tejada et al. (1978); Butler and Locke (1976).
[c] Martin and Dietzmann (1978); Barnes and Summers (1975); Cadle and Mulawa (1978).
[d] Cadle and Mulawa (1978); Gabele et al. (1977).

samples to the absorption apparatus. The absorption method used was similar to that described in EPA Method 8 of the Federal Register for determination of sulfuric acid mist and sulfur dioxide emissions from stationary sources (Fed. Reg. 42(160):41754, 1977). The barium chloranilate method was utilized for analysis of sulfur oxides.

2.3 MOTOR VEHICLE AND AIRCRAFT EMISSIONS

2.3.1 Catalyst-Equipped Vehicle Results

2.3.1.1 Background--To meet automotive exhaust emission standards within the time allowed, U.S. automobile manufacturers in the early 1970's settled on one basic approach to cleaning up their engines--the catalytic converter. From the manufacturers' viewpoint, catalyst technology represented the only approach which had a high probability of reducing emissions to the required levels while at the same time protecting their capital, manpower, and technical investments in the conventional internal combustion engine. The oxidation catalyst or catalytic converter exhaust emission control device was introduced in the 1975 model year. Over 70 percent of the 1975 cars and 85 percent of those in the following years have been catalyst equipped. Although the catalytic converter emission control device decreases regulated gaseous emissions, it increases particulate emissions under certain operating conditions, specifically emission of sulfuric acid, a pollutant of environmental concern (Lee and Duffield 1977). This emission of sulfuric acid is a product of the combustion of fuel sulfur and of catalytic oxidation.

Automotive catalytic converters are composed of small amounts of noble metals, mainly platinum (Pt), palladium (Pd), and rhodium (Rh), either individually or in combination, coated on a support of aluminum oxide. This metal/aluminum oxide catalytic medium is commonly applied as a coating either to a honeycomb ceramic gas contactor (monolithic catalyst) or to aluminum oxide pellets (pelleted catalyst). The finished catalysts are enclosed in stainless-steel containers in a variety of shapes and sizes.

Both types of catalysts, monolithic and pelleted, require approximately the same amount of noble metal, 1-2 g, about the weight of a penny. In a monolithic catalyst, the metal is contained in a very thin layer or "wash coat" of aluminum oxide. Pelleted catalysts, however, contain 2-3 kg of aluminum oxide in the form of spherical beads or cylindrical pellets.

Because of their large alumina content, pelleted catalysts have a much larger capacity than do monolithic catalysts for storing sulfuric acid in the catalyst bed as aluminum sulfate. This factor has an important influence on the sulfuric acid emission process, particularly during vehicle accelerations and decelerations.

2.3.1.2 Mechanism of catalytic sulfate formation--In the combustion process, fuel sulfur is oxidized to sulfur dioxide (SO_2). The exhaust gases, including HC, CO, NO_x, and SO_2, then proceed via the exhaust manifold to the oxidation catalyst. In the catalyst, excess hydrocarbons and carbon monoxide are oxidized to water vapor [$H_2O(g)$] and carbon dioxide, while some of the SO_2 is oxidized to sulfur trioxide (SO_3). Proceeding through the catalyst and into the muffler, the SO_3 combines with $H_2O(g)$ to form gaseous H_2SO_4. This gaseous H_2SO_4 condenses to a finely dispersed aerosol after exiting from the tailpipe, as depicted in Figure 2-3.

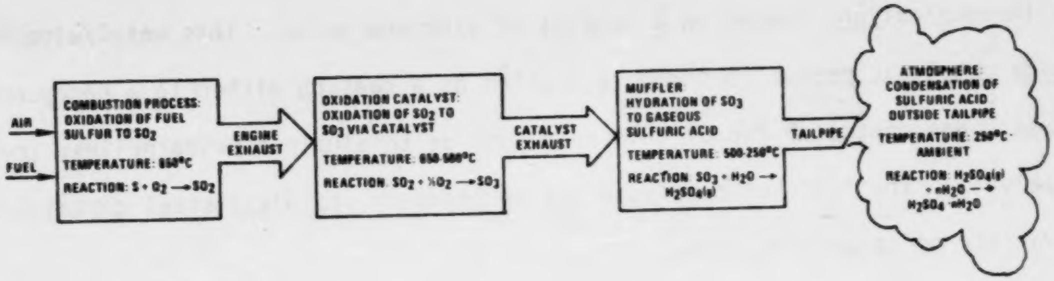
AIR
FUEL
COMBUSTION PROCESS: OXIDATION OF FUEL SULFUR TO SO_2
TEMPERATURE: 850°C
REACTION: $S + O_2 \rightarrow SO_2$
ENGINE EXHAUST
OXIDATION CATALYST: OXIDATION OF SO_2 TO SO_3 VIA CATALYST
TEMPERATURE: 650-500°C
REACTION: $SO_2 + \frac{1}{2}O_2 \rightarrow SO_3$
CATALYST EXHAUST
MUFFLER HYDRATION OF SO_3 TO GASEOUS SULFURIC ACID
TEMPERATURE: 500-250°C
REACTION: $SO_3 + H_2O \rightarrow H_2SO_{4(g)}$
TAILPIPE
ATMOSPHERE: CONDENSATION OF SULFURIC ACID OUTSIDE TAILPIPE
TEMPERATURE: 250°C - AMBIENT
REACTION: $H_2SO_{4(g)} + nH_2O \rightarrow H_2SO_4 \cdot nH_2O$

Figure 2-3. Schematic of sulfur oxidation reactions throughout the exhaust system.

The quantity of SO_3 formed is influenced by many factors, including the concentration of O_2 in the exhaust, the mass flow of sulfur through the engine, the type of catalyst, the temperature of the catalyst, and the possible reactions of SO_2 and SO_3 with other exhaust constituents. In addition, the quantity of H_2SO_4 emitted is affected by sulfur oxide storage and release within the catalyst.

If complete conversion of sulfur dioxide to sulfate were to occur, the average rate of vehicular emission of sulfuric acid would be about 180 mg/mile. The extent to which sulfur dioxide can be oxidized to sulfate is governed by both thermodynamic and kinetic limitations. In the exhaust system, low temperatures and high oxygen levels thermodynamically favor this conversion. However, because of kinetic factors, even under the most favorable laboratory conditions, the theoretical conversion limit is rarely attained. The rate of reaction of SO_2 to SO_3 to $SO_4^=$ is too slow for complete oxidation to occur during the time the exhaust is in the catalyst. Once the exhaust leaves the catalyst, the conversion of SO_2 to SO_3 is minimized. These factors help to explain why observed sulfuric acid emission rates are often much lower than the theoretical limit.

Both kinetic and mass transfer factors indicate that sulfuric acid emission may vary by catalyst type; i.e., some types are inherently more active in produc sulfuric acid than others. In fact, Gabele (1976) showed in steady-state engine dynamometer experiments that Pt/Rh monolithic catalysts form about one-half the sulfuric acid formed by monolithic Pt/Pd catalysts. Beltzer et al. (1977) have reported similar findings in rating various catalysts on a single car, Krause et al. (1976) have reported the same result in a fleet of passenger cars equipped with a variety of catalysts, and Hammerle and Truex (1976b) have reported similar results after testing three different catalyst formulations in a flow reactor utilizing simulated exhaust gases. Thus, it appears possible to construct catalys with potentially low sulfate-forming characteristics.

Gaseous sulfur oxides can react with iron in engines and exhaust systems and with alumina in catalysts to form sulfate or sulfite salts (Knietsch 1901; Krause et al. 1968; Levy et al. 1970). Both iron and aluminum sulfates decompose at temperatures greater than about 425-450°C to produce the corresponding metal oxides and SO_3, the major sulfur-bearing product (Warner and Ingraham 1960, 1962; Kelly et al. 1949; Truex et al. 1977). This mechanism appears to be important in the cyclic storage and release of sulfur oxides, a phenomenon frequently reported with catalyst-equipped passenger cars.

For example, Begeman et al. (1974) found that pelleted-catalyst cars emitted considerably less sulfur (in the form of exhaust SO_2 and H_2SO_4) than the amount present in fuel used during low-speed driving. When these cars were then operated at high speed, SO_2 and H_2SO_4 emissions increased to as much as twice the sulfur then being charged to the engine. Begeman et al. concluded that substantial amounts of H_2SO_4 are stored on pelleted catalysts and that high-speed, high-temperature operation releases this stored material primarily as SO_2. Gibbs et al. (1977) have subsequently reported that exceptionally high transient emissions of SO_2 can be routinely observed in tests of in-use catalyst-equipped cars, particularly during deceleration-to-idle conditions.

Hammerle and Truex (1976a) have reported a number of studies investigating the reductive release of SO_2 from sulfate. Apparently, CO, H_2, and unburned HC can react either with stored sulfate on catalyst beds or with gaseous H_2SO_4 to produce SO_2. This phenomenon accounts for substantially lower sulfuric acid emission rates measured under cyclic driving conditions, especially when compared with those measured under constant-speed driving conditions. Temporary enrichment of the fuel/air mixture fed to the engine during acceleration gives rise to moderate levels of gaseous reducing agents in the catalyst container, and rapid reduction of stored sulfate then occurs, quickly purging the catalyst bed of stored sulfate.

In summary, catalytic oxidation of exhaust sulfur dioxide to form sulfuric acid is subject to both thermodynamic and reaction-rate (kinetic) limitations. In automotive catalysts, sulfuric acid (or sulfur trioxide) may be stored as aluminum sulfate at low catalyst temperatures, only to be thermally and reductively purged as SO_2 under both acceleration and deceleration driving modes.

2.3.2 Diesel-Powered Vehicles

Light-duty diesel-powered passenger vehicles, which are not currently equipped with catalysts, have sulfate emissions comparable to those obtained from some 49-

State gasoline-powered catalyst-equipped vehicles and greater than those obtained from non-catalyst or three-way catalyst gasoline-powered vehicles. Heavy-duty diesel vehicles produce by far the highest vehicular sulfate emissions, well above those produced by most cars equipped with oxidation catalysts. Three factors lead to these higher emissions: (1) diesel fuel contains considerably more sulfur than does gasoline; (2) the oxygen levels of diesel systems are high; and (3) diesel exhaust temperatures are low, which thermodynamically favors the formation of sulfates.

Braddock and Bradow (1975) reported sulfur dioxide and sulfate emissions from a 3500-lb sedan powered by a Nissan naturally aspirated, indirect-injection, light-duty diesel engine. Three different diesel fuels, No. 1, No. 2, and No. 2-D, representing fuel sulfur levels of 0.12 to 0.32 percent by weight, were tested in this study over the FTP and HWFET driving cycles. Depending upon the sulfur content of the fuel, FTP sulfate emissions ranged from 6 to 10 mg/mile, and HWFET sulfate emissions ranged from 10 to 22 mg/mile.

In another study, Braddock and Gabele (1977) reported COS, SO_2, and sulfate emissions from a 1975 Peugeot 504D light-duty passenger vehicle. Four different diesel fuels, Jet A, Local No. 1, National average No. 2, and No. 2-D, representing fuel sulfur levels of 0.04 to 0.29 percent by weight, were tested in this study over the FTP, HWFET, and CUE driving cycles. Using the National average No. 2 diesel fuel (0.23 percent sulfur content by weight), FTP sulfate emissions averaged 8.9 mg/mile, HWFET sulfate emissions averaged 6.4 mg/mile, and CUE sulfate emissions averaged 6.5 mg/mile. COS emissions were relatively low, averaging 0.6 mg/mile during the FTP and 0.2 mg/mile during the HWFET and CUE driving cycles. The majority (>95 percent) of fuel sulfur was emitted as SO_2; with only a minor but important fraction (<3 percent) emitted as sulfate. SO_2 and sulfate emissions increased linearly with increasing fuel sulfur and were greater in the FTP than in either the HWFET or CUE driving cycle.

Springer and Stahman (1977a) reported SO_2 and sulfate emissions from five light-duty diesel-powered passenger vehicles, a Mercedes 220D, a Mercedes 240D, a Mercedes 300D, Peugeot 204D, and a Perkins 6-247. Using National average No. 2 diesel fuel (0.23 percent sulfur content by weight), FTP sulfate emissions averaged 14.5 mg/mile, HWFET sulfate emissions averaged 15.5 mg/mile, and CUE sulfate emissions averaged 13.3 mg/mile. The majority of fuel sulfur (~94 percent) was emitted in the form of SO_2, with ~2 percent emitted in the form of sulfate.

Pierson and McKee (1978) recently reported the results of a test to observe the effect of a catalyst system on sulfate emissions from a light-duty diesel vehicle. Emissions of 6 mg/mile were obtained. Another study, by Marshall et al. (1978), measured sulfate emissions from a catalyst-equipped diesel engine operated on a stand at steady state. Under road load conditions, sulfate emissions averaged 10 to 15 mg/mile. The range of sulfate emissions between 20 and 60 mph was roughly 5 to 50 mg/mile $SO_4^{=}$.

2.3.2.2 Heavy-duty vehicles--Springer and Stahman (1977b) studied diesel sulfate emissions from five different configurations of engines used in heavy-duty vehicles. These configurations included: a Detroit Diesel 8V-71TA truck engine; a Cummins NTC-290 truck engine, which was tested with and without variable injection timing; and a Detroit Diesel Allison Division 6V-71 city bus engine, which was fitted and tested with two different injector designs. The truck engines were operated on DF-2 fuel, and the bus engine was operated on DF-1 fuel. Exhaust measurements showed that 0.9 to 4.5 percent of the sulfur contained in these fuels was converted to sulfate. This range of conversion varied with engine configuration and test load. The average rate of sulfate conversion was 2.5 percent. Sulfur dioxide made up the remainder of the fuel sulfur emitted in exhaust.

2.3.3 Aircraft Gas Turbine

Under contract with EPA, the Pratt & Whitney Aircraft Group of United Technologies Corporation studied emissions from a Pratt & Whitney Corporation PT6A-45 gas turbine aircraft engine (Elwood and Robertson 1978). The PT6A-45, a 1174-horsepower unit widely used in small commercial aircraft, was run at four power settings (idle, approach, climb, and takeoff) with Jet A-1 fuel containing 0.007 percent sulfur by weight, and at three power settings (idle, approach, and climb) with Jet A-1 fuel doped to 0.260 percent sulfur concentration by weight.

The results (see Table 2-6) indicated that no sulfur oxides were detected in the low-sulfur idle and approach test modes. Sampling method limitations, i.e., line absorption losses, probably accounted for the absence of sulfur oxides at these low power settings. The high-sulfur fuel tests did not suffer this limitation, and essentially all the sulfur expected was detected in analysis. As anticipated, sulfur oxides increased in concentration with increasing power requirements. Takeoff, representing 100 percent power, exhibited the greatest SO_x emission with the low-sulfur fuel. Unfortunately, due to combustor temperature limitations, the takeoff test mode was not attainable with the high-sulfur

Table 2-6. PT6A-45 GAS GENERATOR SULFUR OXIDE EMISSION RESULTS IN SULFUR CONVERSION PERCENTS

	Test mode			
	Idle	Approach	Climb	Takeoff
Low-sulfur fuel[a] (0.007 wt% sulfur)				
SO_2	ND[b]	ND	0.0035	0.0179
SO_3	ND	ND	ND	ND
Total SOx	ND	ND	0.0035	0.0179
High-sulfur fuel (0.260 wt% sulfur)				
SO_2	0.2279	0.2769	0.2638	Test not run
SO_3	0.0104	ND	0.0294	Test not run
Total SOx	0.2383	0.2769	0.2932	Test not run

[a] Additional analyses confirmed the 0.007 percent concentration.
[b] ND = None detected.

fuel. Sulfur trioxide or sulfuric acid (i.e., SO_3) was not detected in the low-sulfur fuel tests. It was detected, however, in the high-sulfur fuel runs in the idle and climb tests modes, representing from 4 to 10 percent of the total emitted sulfur oxides [i.e., $(100 \times SO_3)/(SO_2 + SO_3)$].

2.4 EMISSIONS STUDIES

2.4.1 Sulfate Emissions - Introduction

Although there have been numerous experimental studies of sulfate emissions from laboratory-maintained vehicles equipped with catalysts in the past 5 years, there are only two recent dynamometer studies of sulfate emissions from vehicles actually used by consumers. In the first study, Olson Laboratories, Inc., examined gaseous and sulfate emissions as a function of mileage accumulation in 100 automobiles equipped to meet California's emission standards (Herling et al. 1977). The second study, performed by the New York State Department of Environmental Conservation, examined gaseous and sulfate emissions as a function of mileage accumulation in 49 vehicles equipped to meet the emissions standards of the other 49 States (Gibbs et al. 1977). Both studies made use of catalyst-equipped automobiles produced in the 1975-76 model years. A third dynamometer study, the EPA Automotive Sulfate Emissions Baseline Experiment, examined sulfate emissions from 78 vehicles (Somers et al. 1977), including 4 production vehicles without catalysts, 37 production vehicles with catalysts and grouped according to emissions standards, 12 advanced prototype vehicles without catalysts, 20 advanced vehicles with catalysts, and 5 fleet vehicles. The present report will examine only those production vehicles from the EPA Baseline Program that compare directly with those vehicles tested in the Olson Labs and New York State studies. Finally, a study performed by Ford Motor Company will be discussed. This study, unlike the others, measured vehicular sulfate emissions on the road (Pierson et al. 1978). Here, sulfur dioxide and sulfate emissions data were collected in two tunnels on the Pennsylvania Turnpike and reported according to vehicle type.

2.4.1.1 Olson study--The vehicles studied by Olson were certified by their manufacturers to be in conformance with the 1975-76 California emissions standards. All 100 vehicles were privately owned and maintained. Fifty of the cars had monolith catalysts with air injection. The other 50 cars had pelleted catalysts, 35 of which also had air injection. Each vehicle was tested at least three times at intervals of not less than 3 months over a mileage accumulation schedule of approximately 10,000 miles. During idle emissions screening tests, only those vehicles that when idling had CO emissions less than 0.5 percent by volume and HC emissions of less than 50 ppm were accepted into the test program. Approximately 15 percent of the prospective test vehicles failed this initial inspection and were not accepted. Once a vehicle was accepted, however, it was retained in the test fleet regardless of its idle emissions in subsequent testing. Table 2-7 describes the Olson test fleet.

The fuel used in the vehicle tests was the unleaded gasoline normally purchased by the owner. Analysis of the gasoline samples over the entire fleet test program indicated an average fuel sulfur content of 0.027 percent by weight.

Table 2-8 reports the results of this study in terms of average emission rates measured at specific mileage intervals. In reviewing these data, a deterioration in HC and CO emissions between the first and third tests may be noted.

Table 2-7. OLSON STUDY: TEST FLEET DESCRIPTION

Number of vehicles	Manufacturer	Catalyst type	Initial idle emission screening	1975-76 California emission standards	Test schedule[a]
100 total	GM, 44 AMC, 4	Pelleted, 50 (35 with air injection)	HC ≤ 50 ppm	HC = 0.9 g/mile	Conditioning hot LA-4 and overnight soak
74 1975's	Toyota, 2			CO = 9.0 g/mile	
26 1976's			CO ≤ 0.5%	NOx = 2.0 g/mile	1. FTP 2. CUE 3. CUE 4. HWFET 5. CUE 6. CUE
All are consumer-owned vehicles	Ford, 26 Chrysler, 20 Datsun, 2 VW, 2	Monoliths, 50 (all with air injection)			

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[a] Five-minute idles between tests.

Table 2-8. OLSON STUDY: TEST FLEET AVERAGE RESULTS[a]

Test	Mileage	Median FTP HC, g/mile	Median FTP CO, g/mile	Median FTP NOx, g/mile	Average CUE % sulfate conversion rate	Average CUE sulfate emission rate in mg/mile normalized to 0.030 wt% sulfur fuel[b]
I	3,395	0.44	4.27	2.21	10.2	18.2
II	7,360	0.47	5.15	2.11	8.3	14.9
III	11,571	0.47	5.09	1.96	6.0	10.7

[a] The test schedule was as follows: (1) hot conditioning LA-4 followed by a 12- to 20-hr overnight soak; (2) FTP; (3) CUE; (4) CUE; (5) HWFET; (6) CUE; (7) CUE. Tests were separated by 5-min idle periods. All testing and procedures followed standard EPA specifications.

[b] Assumes average fleet vehicle has a inertia weight of 4000 lb and achieves 14.6 mpg on the CUE test procedure.

Not only do these emission increase, but the variance of the data also increases as the vehicles accumulate mileage. The CUE percent sulfate conversion rate reported in Table 2-8 was averaged over four CUE test procedures, each of which produced statistically consistent results. Table 2-9 presents average FTP gaseous emission and CUE normalized sulfate emission results by vehicle manufacturer. The Chrysler vehicles, all equipped with UOP (Universal Oil Products) air-injected monoliths, were the most efficient converters of fuel sulfur to sulfate, emitting ~25.1 mg/mile normalized CUE sulfate. Less efficient were the GM pelleted catalysts and Ford monolith catalysts equipped with air injection, both emitting ~15.6 mg/mile normalized CUE sulfate. The Ford vehicles equipped with Matthey-Bishop monoliths and the GM vehicles equipped with pelleted catalysts without air injection produced the lowest CUE normalized sulfate emissions, 2.9 and 4.8 mg/mile, respectively. Overall, the entire fleet emitted an average of 14.6 mg/mile normalized CUE sulfate over the three test periods.

2.4.1.2 New York State Department of Environmental Conservation study--In this study of 49 consumer-owned vehicles, 24 of the vehicles were privately owned, 24 were fleet cars, and one was used as a control. Thirty-one of the cars had monolith catalysts, and 18 had pelleted catalysts. Nine of the 31 monoliths and 2 of the 18 pelleted catalysts had air injection. Table 2-10 describes the test fleet. Vehicles were scheduled for testing at intervals of approximately 5000 miles. Most of the vehicles were tested approximately three times over a 10,000-mile mileage accumulation. Over 90 percent of all testing was performed on vehicles with less than 28,000 odometer miles. Vehicles were not screened before entry to the test group. It was observed that carburetor idle mixture enrichments, perhaps to relieve the stalling and hesitation tendencies of catalyst vehicles,

Table 2-9. OLSON STUDY: INDIVIDUAL MANUFACTURER RESULTS

Classification of tests	No. of cars	Average FTP HC, g/mile	Average FTP CO, g/mile	Average FTP NOx, g/mile	Average CUE % sulfate conversion rate	Average CUE sulfate emission rate in mg/mile normalized to 0.030 wt% sulfur fuel
Entire fleet	100	0.46	4.84	2.09	8.2	14.6
All GM vehicles	44	0.52	5.34	1.91	6.9	12.4
Pelleted with air[a]	29	0.48	5.03	1.83	8.6	15.4
Pelleted w/o air	15	0.58	6.33	1.65	2.7	4.8
All Ford vehicles	26	0.47	4.33	1.97	6.9	12.4
Engelhard	19	0.44	3.90	2.04	8.9	15.9
Matthey-Bishop with air	7	0.73	6.50	1.79	1.6	2.9
All Chrysler vehicles (all UOP monoliths with air)	20	0.39	4.75	3.62	14.0	25.1

[a] Emission results include data from a total of 35 pelleted-with-air vehicles (29 GM cars and 6 non-GM cars).

Table 2-10. NEW YORK STATE DEPARTMENT OF ENVIRONMENTAL CONSERVATION STUDY: TEST FLEET DESCRIPTION

Number of vehicles	Manufacturer	Catalyst type	Initial idle emissions screening	1975-76 Federal emission standards	Test schedule[a]
49 Total	GM, 18	Pelleted, 18 (2 with air injection)	HC, none	HC = 1.5 g/mile	Vehicle check-in and overnight soak
29 1975's			CO, none	CO = 15 g/mile	1. FTP 2. 1-hr, 50-mph steady state
20 1976's	Ford, 9	Monoliths, 9 (all with air injection)	Vehicles not screened before entry to the test group	NOx = 3.1 g/mile	3. CUE 4. CUE 5. CUE 6. CUE 7. CUE
24 Consumer-owned vehicles	Chrysler, 22	Monoliths, 22 (none with air injection)			
24 Fleet vehicles					
1 Control vehicle					

[a] Five-minute idles between tests.

were quite common. In fact, the fleet averaged 2.4 percent CO emission at idle. Twenty-eight percent of the Ford vehicles (all equipped with air injection), 50 percent of the GM vehicles, and 73 percent of the Chrysler vehicles emitted more than 1 percent CO at idle.

The fuel used in the vehicle tests was the unleaded gasoline normally purchased by the individual test participant. Analysis of the gasoline samples over the entire test program indicated an average fuel sulfur content of 0.017 percent by weight.

Averages of the regulated gaseous and sulfate emission rates are reported in Table 2-11 as a function of the listed subclassifications. As in the Olson study, a significant deterioration in HC and CO emissions occurred with mileage accumulation. As these emissions increased, sulfate emissions tended to decrease, with the maximum sulfate emission rate occurring at approximately 8,000-10,000 miles. The fleet's average sulfate emission rate was 4.7 mg/mile.

A review of the data summarized in Table 2-11 showed that when FTP carbon monoxide emission was less than 15 g/mile, the sulfate emission rate increased to 8.6 mg/mile. This rate further increased to 10.2 mg/mile when Federal HC, CO, and NO_x standards were met. There was a direct relationship between idle CO emissions and CO mass emissions, and an inverse relationship between idle CO and sulfate emissions. Fifty-four tests on vehicles with less than 1 percent CO at idle yielded an average mass emission of 10.3 g/mile FTP carbon monoxide and an average sulfate emission rate of 9.0 mg/mile. Seventy-three tests on vehicles having greater than 1 percent idle CO yielded an average 36.0 g/mile FTP carbon monoxide mass emission and an average 1.6 mg/mile sulfate rate. Consequently,

Table 2-11. NEW YORK STATE DEPARTMENT OF ENVIRONMENTAL CONSERVATION STUDY: FLEET AVERAGE RESULTS[a]

Classification of tests	No. of tests	No. of cars	Idle CO, %	Average FTP HC, g/mile	Average FTP CO, g/mile	Average FTP NOx, g/mile	Average CUE sulfate emission rate in mg/mile normalized to 0.030 wt% sulfur fuel
Total fleet	127	49	2.4	1.7	25.1	2.9	4.7
FTP CO < 15 g/mile	53	23	0.4	0.9	7.6	3.2	8.6
FTP CO < 15 g/mile FTP HC < 1.5 g/mile	49	22	0.3	0.8	7.1	3.2	9.2
FTP CO < 15 g/mile FTP HC < 1.5 g/mile FTP NOx < 3.1 g/mile	30	15	0.4	0.7	7.5	2.5	10.2
Idle CO ≤ 1%	54	26	0.2	0.9	10.3	3.0	9.0
Idle CO > 1%	73	34	4.1	2.3	36.0	2.7	1.6

[a] The test schedule was as follows: (1) overnight soak for 12 to 20 hr; (2) FTP; (3) 1-hr, 50-mph steady-state run; (4) CUE; (5) CUE; (6) CUE; (7) CUE; (8) CUE. Tests were separated by 5-min idle periods. All testing and procedures followed standard EPA specifications.

those vehicles that were tampered with (i.e., had their idle mixtures enriched) emitted less sulfate than those vehicles whose idle mixtures were set according to the manufacturers' specifications. Table 2-12 presents average FTP gaseous emission and CUE normalized sulfate results by vehicle manufacturer. The Ford vehicles with air injection exhibited the highest average CUE sulfate emission (15.0 mg/mile) and the lowest average idle CO emission (0.7 percent). The presence of air injection in these vehicles increased both sulfate production and the ability of the converter to operate effectively without proper carburetor adjustment. The GM vehicles equipped with pelleted catalysts exhibited the lowest average CUE emission rate of 2.8 mg/mile, followed closely by the Chrysler vehicles equipped with the UOP monoliths, which exhibited an average CUE emission rate of 3.3 mg/mile. Overall, the fleet had an average CUE emission rate of 4.7 mg/mile, with air-injected monoliths emitting 15.0 mg/mile, and monolithic and pelleted catalysts without air injection emitting ~3.0 mg/mile.

2.4.1.3 EPA Baseline Experiment--In this study, 25 catalyst-equipped vehicles, acquired either through rental agencies or vehicle manufacturer, were carefully maintained according to the manufacturers' specifications by laboratory personnel. Odometer readings ranged from 50 to 16,200 miles. The vehicles were categorized according to emission standards: Category A (11 vehicles) - 1975-76 Federal emission standards; Category B (5 vehicles) - 1977 Federal emission standards; and Category C (9 vehicles) - 1975-76 California emission standards. Table 2-13 describes the test fleet. The vehicles were uniformly preconditioned over 500-1000 miles on fuel containing 0.030 percent sulfur by weight in order to minimize the effects of any previously stored sulfate. The purpose of this study was to obtain sulfate emission factors from a variety of production vehicles and to

Table 2-12. NEW YORK STATE DEPARTMENT OF ENVIRONMENTAL CONSERVATION STUDY: INDIVIDUAL MANUFACTURER RESULTS

Classification of tests	No. of tests	No. of cars	Idle CO, %	Average FTP HC, g/mile	Average FTP CO, g/mile	Average FTP NOx, g/mile	Average CUE sulfate emission rate in mg/mile normalized to 0.030 wt% sulfur fuel
All GM vehicles	50	18	1.8	1.3	16.4	3.0	2.8
All Ford vehicles	18	9	0.7	1.0	11.5	3.2	15.0
All Chrysler vehicles	59	22	3.5	2.2	36.5	2.6	3.3

Table 2-13. EPA BASELINE PROGRAM: TEST FLEET DESCRIPTION

Category and number of vehicles	Manufacturer	Catalyst type	Emission standards classification	Initial idle emissions screening	Test schedule
A: 11 vehicles 10 1975's 1 1976	AMC, 1 GM, 1 Ford, 7 Chrysler, 2	Pelleted, 2 (1 with air injection) Monoliths, 9	1975-76 Federal HC = 1.5 g/mile CO = 15 g/mile NOx = 3.1 g/mile	All vehicles carefully maintained according to manufacturer specifications by laboratory personnel	Extensive vehicle preconditioning on 0.030 wt% sulfur fuel Conditioning LA-4 and overnight soak 1. FTP 2. CUE 3. CUE 4. HWFET 5. CUE 6. CUE
B: 5 vehicles 3 1975's 1 1976 1 1977	AMC, 1 GM, 2 Ford, 1 Chrysler, 1	Pelleted, 3 (2 with air injection) Monoliths, 2 (no air)	1977 Federal HC = 1.5 g/mile CO = 1.5 g/mile NOx = 2.0 g/mile		
C: 9 vehicles 5 1975's 4 1976's	AMC, 1 GM, 5 Ford, 1 Chrysler, 1 VW, 1	Pelleted, 6 (3 with air injection) Monoliths, 3 (2 with air injection)	1975-76 California HC = 0.9 g/mile CO = 9 g/mile NOx = 2.0 g/mile		

assess the effect of sulfate emissions from vehicles meeting increasingly stringent emission standards for HC, CO, and NO_x.

Fleet averages of the FTP gases and CUE sulfate emission rates are reported in Table 2-14 as a function of emission standard classification. In this study, as in the Olson Labs and New York State Department of Environmental Conservation studies, the vehicles were not tested at high mileage. The maximum test mileage for any vehicle in the Baseline Program was 16,200 miles, and the fleet's average odometer reading was ~7000 miles. It was observed that few of the consumer-owned test vehicles in either the Olson Labs or New York State studies met their respective emission standards, while most of the 25 test vehicles in the EPA Baseline Program did meet these standards. The principal reasons for this difference are that the EPA test fleet was small (25 vehicles), had low mileage, and was carefully maintained by skilled laboratory technicians. The 11 vehicles in Category A (1975-76 Federal emission standards) averaged 15.0 mg/mile normalized CUE sulfate, the 5 vehicles in Category B (1977 Federal emission standards) averaged 9.0 mg/mile normalized CUE sulfate, and the 9 vehicles in Category C (1975-76 California emission standards) averaged 15.7 mg/mile normalized CUE sulfate. Although the EPA Baseline Program did not measure gaseous and sulfate emissions as a function of mileage, as did the Olson Labs and New York State studies, it is reasonable to assume that there would be some deterioration of the vehicles' emission control system(s) with mileage accumulation, thereby increasing regulated gaseous emissions (HC, CO, and NO_x) and decreasing sulfate emissions. This decline in sulfate emission has been observed experimentally by Exxon Research and Engineering (Krause et al. 1976) and is currently the subject of an EPA-organized sulfate deterioration factor study. Consequently, the normalized

Table 2-14. EPA BASELINE PROGRAM: TEST FLEET AVERAGE RESULTS[a]

Category and number of vehicles	Average FTP HC, g/mile	Average FTP CO, g/mile	Average FTP NOx, g/mile	Average CUE sulfate emission rate in mg/mile normalized to 0.030 wt% sulfur fuel
A: 11 vehicles (1975-76 Federal emission standards 1.5/15/3.1)	0.60	4.36	2.25	15.0 (Range, 0.5-42.5)
B: 5 vehicles (1977 Federal emission standards 1.5/15/2.0)	0.61	5.04	2.04	9.0 (Range, 2.9-22.4)
C: 9 vehicles (1975-76 California emission standards 0.9/9/2.0)	0.56	8.79	1.62	15.7 (Range, 0.2-52.2)

[a] The test schedule was as follows: (1) conditioning LA-4 followed by a 12- to 20-hr overnight soak; (2) FTP; (3) CUE; (4) CUE; (5) HWFET; (6) CUE; (7) CUE. Methods and procedures followed standard EPA specifications.

average CUE sulfate emission rates listed in Tables 2-14 and 2-15 probably represer upper limits for those vehicles tested in Categories A, B, and C.

In addition, EPA has conducted emission factor studies of 66 consumer-owned vehicles in Denver, 64 consumer-owned vehicles in Houston, 155 consumer-owned vehicles in Phoenix, and 75 consumer-owned vehicles in St. Louis (Carlson 1977; Liljedahl and Terry 1977). The results of these studies indicated that these in-use vehicles had lower sulfate emissions than the EPA Baseline Program would have predicted. The average CUE sulfate emission rate, normalized to fuel containing 0.030 percent sulfur by weight, was on the order of ~5 mg/mile. This sulfate emission rate (~5 mg/mile) compares very favorably with that obtained by the New York State Department of Environmental Conservation (~4.7 mg/mile) for in-use vehicles.

2.4.1.4 Ford Motor Company roadway study--In this study, conducted intermittently over a 3-year period (1974-76), vehicular sulfate emissions were determined by measuring roadway sulfate levels in the Allegheny and Tuscarora tunnels on the Pennsylvania Turnpike. The objectives of this study were to: (1) determine sulfate emission rates from vehicles in actual roadway use, including catalyst-equipped automobiles, automobiles without catalysts, and diesel trucks; (2) rank the respective vehicular sulfate emissions with each other and with ambient sulfate levels; and (3) compare roadway emission rates with emission rates determined from dynamometer studies.

The experiments involved measurement of roadway exhaust emissions of SO_2, sulfate, and two fuel additives: lead (Pb), which is used to improve octane in

Table 2-15. EPA BASELINE PROGRAM: INDIVIDUAL MANUFACTURER RESULTS

Vehicle classification	No. of cars	Average FTP HC, g/mile	Average FTP CO, g/mile	Average FTP NOx, g/mile	Average CUE sulfate emission rate in mg/mile normalized to 0.030 wt% sulfur fuel
All AMC vehicles (all pelleted with air)	3	0.42	5.45	2.17	10.0
All GM vehicles	8	0.55	9.11	1.74	9.1
Pelleted with air	3	0.52	9.05	2.26	11.4
Pelleted w/o air	5	0.57	9.15	1.43	7.7
All Ford vehicles	9	0.65	3.41	2.09	17.2
Monoliths with air	8	0.65	3.47	2.13	19.0
Monoliths w/o air	1	0.66	2.95	1.74	2.9
All Chrysler vehicles	4	0.72	7.04	2.05	22.7
Monoliths with air	1	0.64	9.27	1.67	52.2
Monoliths w/o air	3	0.74	6.29	2.18	12.8
All VW vehicles (monolith w/o air)	1	0.26	4.19	2.03	2.3

gasoline-powered non-catalyst vehicles, and barium (Ba), which is added to diesel fuel to suppress smoke. These emissions were then correlated with the amounts of sulfur, Pb, and Ba contained in the fuels of the various types of vehicles passing through the tunnel. Measurements were made in 1974, when there were no catalyst-equipped vehicles on the road; in 1975, when an estimated 10-15 percent of the vehicles were equipped with catalysts; and in 1976, when an estimated 28 percent were so equipped. These estimates were based on the amounts and types of fuels sold at the turnpike service plazas near the tunnels. Thus, the approximate number of vehicles and the sulfur content of their fuels were obtained according to the date of testing (1974, 1975, or 1976). To identify sources of the sulfate emissions according to vehicle type, multiple regressions of sulfate against Ba and Pb were performed.

The 1976 averages of SO_2 and sulfate emissions in the Tuscarora Tunnel are presented in Table 2-16 according to vehicle type. The normal vehicular driving pattern through the tunnel was to decelerate from 58 mph to 47 mph upon approach and then to maintain an average speed of 47 mph throughout the tunnel. This drive lasts 1.47 min at a steady 47 mph and thus represents a sort of 1/10th-scale HWFET procedure (not a CUE procedure, as used in the three previous studies). Sulfate emission rates for cars without catalysts were on the order of $\sim$1-2 mg/mile; for catalyst-equipped cars, $\sim$8-13 mg/mile; and for diesel trucks, $\sim$50 mg/mile. It is obvious that diesel trucks, representing $\sim$18 percent of the total traffic entering the tunnel and combusting fuel containing 0.20 percent sulfur by weight, were the dominant sulfate emitters, accounting for two-thirds of the total sulfate emitted. Since diesel trucks log $\sim$5 percent of the total vehicle miles nationwide, it follows that they should be responsible for about one-third of vehicle-produced sulfate.

Table 2-16. 1976 TUSCARORA TUNNEL RESULTS

Vehicle type	Average sulfate emission rate in mg/mile normalized to 0.030 wt% sulfur fuel	Average SO_2 emission rate in mg/mile normalized to 0.030 wt% sulfur fuel	$SO_2 \rightarrow SO_4^=$ conversion, %
All traffic	~12.0	356	2.2
All cars	~2.3	84	1.4
Non-catalyst cars	~1-2		
Catalyst cars	~10 (Range, 8-13)		
Diesel trucks (~18% of total vehicle population)	~50	1379	2.4

Table 2-17 presents average ambient and tunnel-measured sulfate and SO_2 concentrations in $\mu g/m^3$. These measurements show that sulfate from vehicles is minor compared with ambient sulfate levels at this location. Despite the tunnel's confinement of vehicular emissions, the tunnel sulfate concentrations in 12-hr samples exceeded the background by 8.3 $\mu g/m^3$ at most, while the background itself measured up to 39.1 $\mu g/m^3$. Thus it appears that even along a rural roadway, vehicular sulfate would be overshadowed by other sulfate sources. Just the opposite is true, however, for tunnel SO_2 emissions, which exceeded background SO_2 levels by a factor of $\sim$15, indicating that along a rural roadway vehicular SO_2 would probably overshadow other SO_2 sources. About 2.2 percent of the SO_2 from all traffic sources was converted to sulfate. This sulfate represented 2-5 percent of the total mass of airborne particulate matter produced by the traffic.

2.4.1.5 Summary: Sulfate emissions from oxidation catalyst-equipped vehicles--Four sulfate studies have been reviewed: (1) the Olson Labs study involving 100 consumer-owned California cars; (2) the New York State Department of Environmental Conservation study involving 49 consumer-owned cars; (3) the EPA Baseline Program study involving 25 laboratory-maintained and -operated production cars; and (4) the Ford Motor Company roadway study involving cars with catalysts, cars without catalysts, and diesel trucks passing through two tunnels along the Pennsylvania Turnpike. The results of these four studies are summarized in Table 2-18. The overall fleet averages for the dynamometer-tested vehicles in the Olson Labs, New York State, and EPA Baseline studies are normalized CUE (Crowded Urban Expressway) sulfate emission rates. The Ford Motor Company fleet average is a normalized turnpike tunnel sulfate emission rate. Strict comparisons between the various studies' fleet average emission rates are not entirely

Table 2-17. AVERAGE TUNNEL AND AMBIENT SULFATE AND SO_2 CONCENTRATIONS ($\mu g/m^3$)

Sulfur compound	Average concentration	Concentration range
Ambient sulfate	14.2	3.7-39.1
ΔSulfate in tunnel[a]	5.5	2.6-8.3
Ambient SO_2	11.5	2.3-25.2
ΔSO_2 in tunnel[a]	161	73-233

[a] Tunnel concentration minus ambient concentration.

Table 2-18. SUMMARY OF SULFATE EMISSION RATES[a] (mg/mile)

	N[b]	Olson Labs: all 1975-76 California catalyst cars	N	New York State: all 1975-76 49-State catalyst cars	N	EPA Baseline: both 1975-77 California and catalyst cars	Ford Motor Co.: all 1975-76 49-State catalyst cars
Overall fleet average	100	~14.6	49	~4.7	25	~14.1 California cars: 15.7 49-State cars: 13.1	~10.5 (Range, 8-13)
All pelleted catalysts	50	12.4	18	2.8	11	9.4	
Pelleted with air	35	15.4	2	13.8	6	10.7	
Pelleted w/o air	15	4.8	16	1.4	5	7.7	
All monolith catalysts	46	17.9	31	6.7	14	17.7	
Monoliths with air	46	17.9	9	15.0	9	22.7	
Monoliths w/o air	0	--	22	3.3	5	8.7	

[a] Normalized to 0.030 wt% sulfur fuel.
[b] N = Number of vehicles tested in that category.

valid, since the number and types of vehicles, vehicle-preconditioning schedules, vehicle-maintenance schedules, test schedules and procedures, and other variables were not identical for all studies. However, because these studies represent the most recent and comprehensive works to date, and because they tend to complement each other, their results are useful for purposes of comparison in this report.

2.4.1.6 Conclusions--

1. Oxidation catalyst-equipped passenger vehicles emit significantly more sulfate (sulfuric acid) than either production or prototype gasoline-fueled passenger vehicles without catalysts.

2. Vehicles manufactured during the 1975-77 production years and equipped to meet Federal emission standards (applied in all States but California) emit from 4.7 to 13.1 mg of sulfate per mile.

3. Vehicles manufactured during the 1975-76 production year and equipped to meet California emission standards emit from 14.6 to 15.7 mg of sulfate per mile.

4. The range of sulfate emissions in both (2) and (3) above depends upon the type of catalyst and how well the car is tuned. In both (2) and (3), pelleted catalysts have lower sulfate emissions than monolith catalysts. Air injection significantly increases emissions from both these catalysts. Thus, in order of decreasing sulfate emissions, the systems are: monolith with air injection; pelleted with air injection; monolith without air injection; pelleted without air injection.

5. Laboratory-maintained production vehicles exhibit higher sulfate emissions than do consumer-owned production vehicles. Deterioration or alteration of the emission control system(s) of consumer-owned/maintained vehicles increases regulated gaseous emissions (i.e., HC, CO, and NO_x) and decreases sulfate emissions from these vehicles.

6. Sulfate emissions appear to decrease with mileage accumulation in consumer-owned/maintained vehicles.

7. From vehicles equipped to meet Federal (49-State) emission standards, roadway-measured sulfate emission rates (8-13 mg/mile) agree fairly well with dynamometer-measured sulfate emission rates (4.7-15 mg/mile).

8. Diesel trucks exhibit the highest measured vehicular sulfate emission rate, ~50 mg/mile.

2.4.2 SO_2 Emission Studies

It has been observed that only a fraction of exhaust SO_2 is catalytically converted to sulfuric acid. The Olson study, for example, showed that, on a fleet-average basis, only 8.2 percent of the fuel sulfur was emitted as sulfate (see Table 2-9). Exhaust SO_2 may either exit the tailpipe unchanged or react with other exhaust gas constituents, the aluminum oxide catalyst substrate material, and/or the iron contained in the exhaust system. If the SO_2 chemically reacts with the aluminum oxide substrate material, then the SO_2 is stored in the converter as SO_3. This storage is a function of both the way the vehicle is driven and the type of catalyst. Transient driving cycles tend to cause storage of SO_3, while prolonged high-speed driving tends to cause release of stored SO_3. Storage and release values tend to be higher for pelleted catalysts, with their larger surface areas per unit mass, than for monolith catalysts. If a steady-state approximation for the storage and release of SO_3 in the CUE driving cycle is assumed (i.e., the fraction of SO_3 being stored is equivalent to the fraction of SO_3 released), then 99 percent of the sulfur-related emissions can be accounted for in the calculation below. This calculation is based upon a 4000-lb vehicle combusting fuel containing 0.030 percent sulfur by weight and achieving 14.6 mpg in the CUE test procedure. The sulfur oxide emissions listed in parentheses represent the weight of material emitted in milligrams per CUE mile. This calculation also assumes that of all the fuel sulfur combusted, ~99 percent enters the catalyst as SO_2 and 1 percent enters in the form of other sulfur species, including sulfides or other reduced sulfur compounds. If 115 mg of SO_2 enters the catalyst:

(a) 9 percent (10 mg) is catalytically oxidized to sulfate and exits the tailpipe as an $SO_4^=$ aerosol (16 mg);

(b) 63 percent (73 mg) passes through the catalyst unchanged and exits the tailpipe as an SO_2 gaseous emission (73 mg); and

(c) 27 percent (31 mg) reacts with the catalyst's alumina substrate to be temporarily stored and later quantitatively released as an SO_2 gaseous emission (31 mg).

The reactions of SO_2 and sulfides with other exhaust gas constituents have been ignored in this scenario. Consequently, the sulfur-related tailpipe emissions from this automobile are: SO_2 = 104 mg/mile (73 mg/mile from initial combustion process and 31 mg/mile from the intra-catalyst storage/release mechanism); H_2SO_4 = 16 mg/mile; other sulfur species, including sulfides = 1-2 mg/mile. A flowchart concerning sulfur-related compound mass emissions and mass balances is shown in Figure 2-4. The mass balances and percent conversion are based upon the initial mass of SO_2 (115 mg) entering the catalyst, and all emissions are in milligrams per CUE mile.

2.4.3. H_2S and COS Emission Studies

It has been observed that some catalyst-equipped vehicles occasionally produce rather objectionable odors. Hydrogen sulfide (H_2S) and carbonyl sulfide (COS) emissions are primarily responsible for these odors, which usually occur during cold starting and/or hot idling. These odors are especially noticeable when the vehicle's exhaust is confined to a relatively small area, such as in a garage or in dense, slow traffic. Because H_2S has one of the lowest known odor thresholds, ~0.005 ppm (Warner 1976), even small amounts are noticeable to bystanders and the vehicle's occupants. The odor of COS is similar to that of H_2S, although its threshold has not been determined (Peyton et al. 1976).

Barnes and Summers (1975) reported that three conditions favored the formation of H_2S by Pt/Pd oxidation catalysts: (1) rich air/fuel ratio (i.e., a reducing condition); (2) low exhaust space velocity; and (3) high catalyst temperature. These conditions rarely occur with properly tuned vehicles. However, malfunctioning vehicles or vehicles with maladjusted carburetors that run rich (>1.0 percent CO at idle), such as those listed in Table 2-11, may meet these conditions and emit H_2S and COS. It should be noted that the reducing conditions that favor sulfide formation do not favor sulfate formation. Cadle and Mulawa (1978) recently tested four malfunctioning customer-owned, catalyst-equipped vehicles for H_2S and COS emissions during cold starting and hot idling.

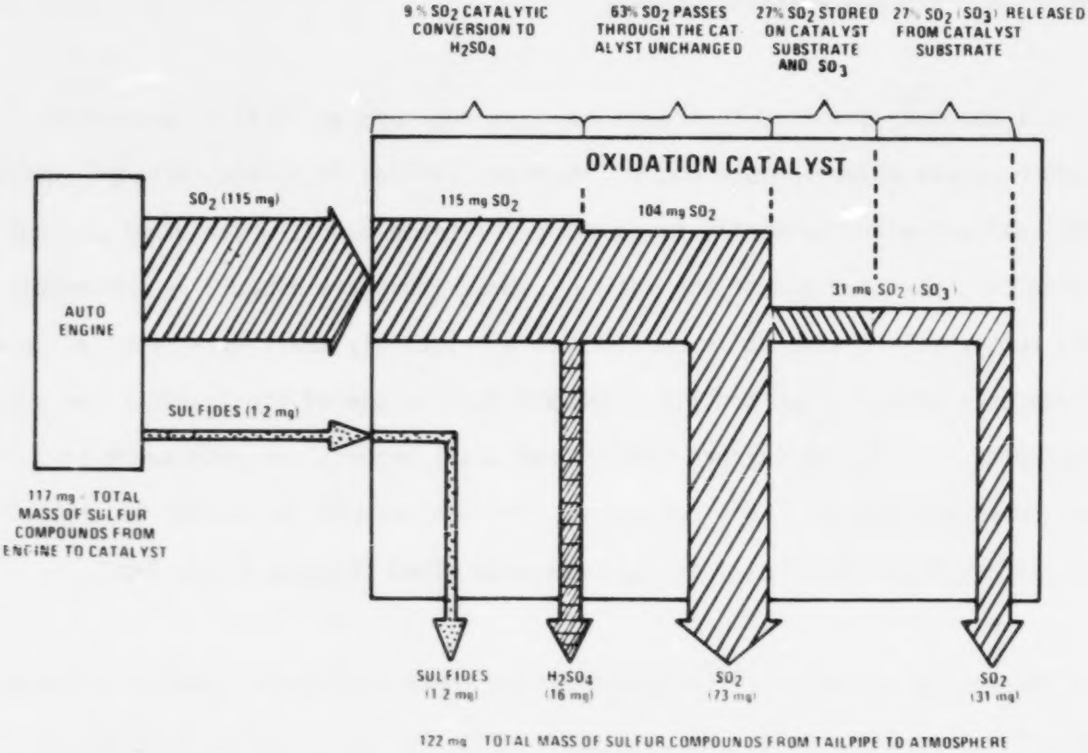
9% SO2 CATALYTIC CONVERSION TO H2SO4
63% SO2 PASSES THROUGH THE CATALYST UNCHANGED
27% SO2 STORED ON CATALYST SUBSTRATE AND SO3
27% SO2 (SO3) RELEASED FROM CATALYST SUBSTRATE
OXIDATION CATALYST
AUTO ENGINE
SO2 (115 mg)
115 mg SO2
104 mg SO2
31 mg SO2 (SO3)
SULFIDES (1.2 mg)
117 mg = TOTAL MASS OF SULFUR COMPOUNDS FROM ENGINE TO CATALYST
SULFIDES (1.2 mg)
H2SO4 (16 mg)
SO2 (73 mg)
SO2 (31 mg)
122 mg = TOTAL MASS OF SULFUR COMPOUNDS FROM TAILPIPE TO ATMOSPHERE

Figure 2-4. Flowchart showing sulfur-related compound mass emissions per CUE mile.

The test vehicles were known to produce odors characteristic of reduced sulfur compounds and were selected on this basis. A special test procedure was developed to maximize sulfide formation and determination. Each vehicle was stored for at least 17 hr at -29°C and then pushed onto a dynamometer in an area where the ambient temperature was 23°C. The vehicle was started and allowed to idle for several minutes. Subsequently, several 10-min cruises at 50 mph were alternated with 10-min idling periods. Both H_2S, in the concentration range of 0.7-6.6 ppm, and COS, in the concentration range of 0.03-1.7 ppm, were detected during this test procedure. These were maximum concentrations which occurred only briefly. The characteristic "rotten egg" odor of H_2S is noticeable at the part per million level. Thus, the maximum emission levels can be detected by persons on or near the roadway. Adverse health effects from exposure to this compound, however, have occurred only at concentrations orders of magnitude higher than the maximum mobile source emissions levels.

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3. HEALTH AND WELFARE EFFECTS

A detailed account of the effects on the public health and welfare from sulfur-bearing compounds emitted from mobile sources can be found in Appendixes A and B; this chapter summarizes the appendix material. The effects on health are discussed first (see Appendix A), followed by the effects on welfare--vegetation, nonbiological materials, and visibility and climate (Appendix B). All cited literature and references appear in the appendixes.

3.1 HEALTH

The general topic of sulfur oxides and sulfates, the primary sulfur-bearing compounds emitted from mobile sources, raises more debate perhaps than any other area of environmental medicine. For years, investigators have sought to delineate the health effects from exposure to these compounds, focusing on sulfur dioxide (SO_2), bisulfite (HSO_3^-), sulfuric acid (H_2SO_4), and sulfate salts ($SO_4^=$). The research has included four major experimental approaches: laboratory studies *in vitro*, animal studies *in vivo*, human clinical studies, and epidemiological studies.

3.1.1 *In Vitro*

In vitro studies are conducted on microorganisms, isolated cells, subcellular fractions, or fluids of animals or man. Such studies help to describe mechanisms of pollutant actions under controlled conditions. This information sometimes serves to explain findings from *in vivo* studies and aids in the development of approaches to in vivo experimentation. In vitro tests may facilitate ranking of pollutants for future in vivo assays. However, the in vitro approach has several limitations. First, the pollutant exposure, particularly in terms of concentration, is usually far removed from natural conditions. In vitro systems may not reflect the transport, clearance, and possible transformation effects occurring in intact animals. Hence, whether concentrations in in vitro exposure mimic those found in in vivo exposure cannot be predicted with certainty. A second major limitation of in vitro test systems is that they are isolated from normal physiological mechanisms, including repair mechanisms which might modify the effect of pollutant exposure in vivo. For these reasons, the results from in vitro studies cannot in themselves serve as a basis for regulatory action. In a supportive role, however, these studies offer an important contribution to health effects research.

Sulfur dioxide is a highly water-soluble gas which hydrates rapidly in solution. Accordingly, when SO_2 is inhaled, it is absorbed in the moist tissue present in the nose or mouth and forms bisulfite ions. Using an artificial tracheobronchial system, researchers found that SO_2 was absorbed primarily in the upper third of a simulated airway, while only a small fraction reached a simulated alveolar and respiratory bronchial region. In in vivo systems, the bisulfite ion formed upon absorption is probably carried up the bronchial tree and subsequently ingested.

In a study in which SO_2 was combined with an aerosol of sodium chloride (NaCl), the SO_2 was deposited in the more distal parts of a simulated lung. This action has also been demonstrated in vivo. Since small (submicron) particles penetrate deeply into the airways, SO_2 absorbed on these particles penetrates more deeply than does SC_2 alone.

In studies using the isolated rat lung, sulfate transport and absorption were found to vary inversely with particle size. The cation associated with the sulfate was also an important factor. For instance, absorption was greater with ammonium or several metal sulfates than with manganese sulfate ($MnSO_4$). In studies with guinea pigs, $MnSO_4$ had no effect on pulmonary function, whereas ammonium and some other metal sulfates did.

Sulfate salts cause histamine release from lung fragments *in vitro*. This release may be a mechanism of bronchoconstriction, which has been noted in *in vivo* studies. Sulfates also stimulate the production of a superoxide radical, which hypothetically may be responsible for pulmonary inflammation, a symptom thought to underlie many of the chronic lung diseases seen in man.

Sulfur dioxide reacts with all major classes of biomolecules, including nucleic acids and proteins. In its hydrated form as bisulfite, its mutagenicity has been demonstrated in bacteria, viruses, yeast, and cultured mammalian cells. Whether bisulfite is mutagenic to mammalian cells *in vivo* is not yet known. To have this effect, enough bisulfite would have to be present in a form capable of penetrating the cytoplasm or nucleus of the cell. To counter this exposure, protective mechanisms can sequester bisulfite until it is excreted from the body or convert it to a less toxic sulfate species.

A summary of comparisons between in vitro and in vivo studies of sulfur-bearing compounds is presented in Table 3-1.

Table 3-1. COMPARISONS BETWEEN IN VITRO AND IN VIVO STUDIES OF SULFUR-BEARING COMPOUNDS

Chemical	Effects: In vitro	Effects: In vivo
$Zn(NH_4)_2(SO_4)_2$	Changes in alveolar macrophages	Increased susceptibility to disease in mice
$SO_4^{=}$	Histamine release from lung fragments	Bronchoconstriction
SO_2/HSO_3^-	Mutagenesis in cultured mammalian cells	With BP, pulmonary carcinogenesis in rats
$MnSO_4$, Na_2SO_4	No substantial histamine release	No bronchoconstriction
$MnSO_4$	No enhanced absorption with manganese cations	No effects of pulmonary mechanics
$(NH_4)_2SO_4$	Marked increase in histamine release	Bronchoconstriction
$SO_2 + NaCl$	Deposited to more distal parts of a simulated lung	Increased changes in pulmonary mechanics
$SO_2 + CuSO_4$	Divalent copper readily forms sulfite complexes	Caused more than an additive effect on pulmonary function
SO_2/HSO_3^-	Increased ATPase activity in rat alveolar macrophages	Increased ATPase and lysozyme activity in baboon alveolar macrophages
SO_2/HSO_3^-	SO_2 absorbed in a simulated tracheobronchial system	In most animals, >90% SO_2 is absorbed in upper airways
SO_2/HSO_3^-, SO_4	Greater amounts of histamine release in guinea pigs	Higher sensitivity to SO_2 + $SO_4^{=}$ aerosols in guinea pigs

3.1.2 In Vivo

In vivo animal studies allow examination of a wide range of concentrations, exposure regimens, and chemical agents and provide data on many of the effects of SO_x, such as morphological alterations and dysfunction of host defense mechanisms against infectious disease. Animal studies help to predict the likelihood that observed effects will occur in humans, but they cannot establish with certainty at what levels of exposure such effects will occur.

Most *in vivo* work has utilized short-term exposures to SO_2 and $SO_4^=$. These studies have been limited, for the most part, to pollutant effects on pulmonary function. Vast areas remain to be explored--long-term exposure regimens, the impact of other sulfur-bearing compounds present in the atmosphere, and the various other physiological parameters that may be affected. The following discussion summarizes many animal exposure studies that have been conducted to date.

Sulfur dioxide is an upper respiratory tract irritant absorbed primarily in the upper airways. In rabbits exposed to concentrations as high as 100 ppm (26,000 $\mu g/m^3$), about 95 percent of the sulfur dioxide was absorbed in the upper airways and thereby prevented from reaching the deep lungs. Subsequently an experiment using radiolabeled sulfur dioxide disclosed that after absorption the compound reached the lung via the bloodstream.

Shortly after inhalation, sulfur dioxide causes a narrowing of the bronchial tubes, resulting in an increase in airway resistance. A 1-hr exposure to as little as 0.5 ppm (1300 $\mu g/m^3$) increases pulmonary flow resistance in guinea pigs. Generally concentrations greater than 1 ppm (2600 $\mu g/m^3$) increase pulmonary flow resistance and respiratory rates in various animals. It has also been noted that young animals appear to be more sensitive than adult animals.

The effects of long-term exposures to sulfur dioxide vary with concentration, duration, and animal species. Generally, relatively high concentrations (far above prevailing ambient levels) are required to produce a chronic response such as alteration of pulmonary structures and ventilatory function. For example, changes in the respiratory mechanics of dogs were observed after 225 days of continuous exposure to 5 ppm SO_2. These changes included increased pulmonary flow resistance and decreased lung compliance (a measure of lung distensibility).

The sulfuric acid contained in an aerosol is particulate matter, and therefore its toxicity depends to some extent on its deposition and retention in the respiratory tract. The acid is considerably more potent than sulfur dioxide in exerting physiological effects. One recent study, for example, showed that sulfuric acid more than tripled the increase in pulmonary flow resistance caused by an equimolar amount of SO_2.

The sulfuric acid mist produced by mobile sources disperses rapidly into dilute suspensions and reacts with ions in the atmosphere, such as ammonia, to form atmospheric sulfate salts. On the basis of studies with guinea pigs, sulfuric acid and various sulfate salts have been ranked according to physiological response on a scale of 1 to 100.

Sulfuric acid	100	Ammonium bisulfate	3
Zinc ammonium sulfate	33	Copper sulfate	2
Ferric sulfate	26	Ferrous sulfate	0.7
Zinc sulfate	19	Sodium sulfate	0.7
Ammonium sulfate	10	Manganese sulfate	-0.9*

*Not significant.

Among the factors determining sulfate toxicity are the cationic species, particle size, and hygroscopicity. It has also been noted that foreign nuclei (such as salts) in hygroscopic vapors may affect the size and hence the toxicity of the aerosol.

Short-term exposure to 70-100 mg/m^3 sulfuric acid causes an increase in pulmonary flow resistance in guinea pigs. These animals appear to be the most sensitive species to sulfuric acid, followed by mice, rats, and rabbits. For example, exposures to 100 mg/m^3 sulfuric acid for 2 hr are fatal to guinea pigs, while the same concentration is not fatal to rats, even after a 6-month exposure.

Recent studies seem to bring together much of the previous work on sulfate salts, including the relation between their toxicity and particle size. In guinea pigs, smaller particles produce greater changes in pulmonary resistance at a given concentration than do larger particles. The response to 0.1 mg/m^3 $SO_4^=$, 1-μm particle size, is slight and rapidly reversible, while the same concentration with 0.3-μm particles is greater in magnitude and not rapidly reversible. Generally, the response per microgram of sulfate ion increases as particle size decreases.

Studies of long-term continuous exposure of dogs and monkeys to 0.8-5.0 mg/m^3 sulfuric acid have shown changes in lung volumes and pulmonary structures, decreases in heart weight, and elevations in total pulmonary resistance, while a long-term study using 0.1 mg/m^3 has shown no significant effects.

Generally, the differences noted between short-term exposures to high doses and chronic exposures to low doses of H_2SO_4 might be explained by the presence of ammonia in the upper airways. It has been suggested that ammonia may mitigate the effects of inhaled acid sulfate by neutralization, particularly at low concentration levels.

When combined with a sodium chloride aerosol, SO_2 exerts a greater effect on pulmonary mechanisms than SO_2 alone. As noted previously, this combination penetrates into the distal parts of the lung. Guinea pigs exposed to SO_2 were found to show no additional effects when the SO_2 was combined with various ammonium sulfate salts. However, the combination of SO_2 with copper sulfate, by itself one of the less potent salts, produced more than an additive effect. Since divalent copper readily forms sulfite complexes, this observed effect may have resulted from the formation of a copper sulfite aerosol. The combination of ozone and sulfur dioxide produced responses similar in degree and mechanics to that of O_3 alone, indicating no interactive effects from this combination.

The order of exposure to pollutant combinations may also affect pollutant toxicity. For example, in one study mice were exposed to H_2SO_4

and/or ozone, followed by a bacterial challenge. Exposure to either the acid or ozone alone had no effect on mortality, nor did a sequential exposure first to the acid and then to ozone. However, when the animals received sequential exposure to the ozone first, significant mortality occurred.

Several long-term studies have involved SO_2/H_2SO_4 combinations. Two of these studies measured pulmonary function 2 to 3 years after a long-term experiment in which dogs were exposed to irradiated and nonirradiated auto exhaust with or without SO_2 and H_2SO_4 singly and combined. Flow resistance was elevated and total lung capacity was increased in all animals except those that had breathed filtered air.

The carcinogenicity from inhaling a combination of two urban air pollutants, SO_2 and benzo(a)pyrene (BP), was studied in rats and hamsters. No significant pathology was found in hamsters, but squamous cell metaplasia and carcinoma were noted in rats exposed to the BP/SO_2 combination. The incidence of metaplasia and carcinoma increased when a separate SO_2 exposure was administered with the above combination. No data were given for exposure to BP alone. A similar study using BP/H_2SO_4 combinations is being conducted by the U.S. Environmental Protection Agency.

A summary of the dose effects of sulfur-bearing compounds on various animal species is presented in Table 3-2.

Table 3-2. DOSE EFFECTS OF SULFUR-BEARING COMPOUNDS ON VARIOUS ANIMAL SPECIES

Chemical	Concentration, mg/m^3	Time	Species	Effect
H_2SO_4	0.1-1.0	30 min	Guinea pig	Increased pulmonary resistance
H_2SO_4	1	2 hr	Hamsters Rabbits	Decreased ciliary activity; hematological changes, but no immune changes
H_2SO_4	2.4 and 4.8	2 hr	Monkeys	Pulmonary function changes and alteration of lung structure
$ZnSO_4$ or $Zn(NH_4)_2(SO_4)_2$	1.3 or 2.1	3 hr	Mice	Increased susceptibility to infection (dose-response effects)
$(NH_4)_2SO_4$	1-$\geq$4.1	3 hr	Mice	No increased susceptibility to infection
SO_2 with and without carbon particles	5.2	102 days 192 days	Mice	Changed immune system
H_2SO_4 on carbon particles	1	3 hr/day, 5 days/wk, 20 wk	Mice	Altered immune system and lung structure
$O_3 + H_2SO_4$	0.2 + 1.0	3 hr + 2 hr	Mice	Increased susceptibility to infection
$Zn(NH_4)_2(SO_4)_2$	1.1	1 hr	Guinea pig	81% increase in pulmonary resistance
$ZnSO_4$	0.9	1 hr	Guinea pig	40% increase in pulmonary resistance
NH_4SO_4	1.0	1 hr	Guinea pig	22% increase in pulmonary resistance
$FeSO_4$	1.0	1 hr	Guinea pig	77% increase in pulmonary resistance
$Fe_2(SO_4)_3$	1.0	1 hr	Guinea pig	2% increase in pulmonary resistance

Table 3-2 (Continued). DOSE EFFECTS OF SULFUR-BEARING COMPOUNDS ON VARIOUS ANIMAL SPECIES

Chemical	Concentration, mg/m^3	Time	Species	Effect
$MnSO_4$	4.0	1 hr	Guinea pig	No increase in pulmonary resistance
SO_2 + NaCl	0.41-26.0	1 hr	Guinea pig	Increased pulmonary resistance, tidal volume, and frequency of breathing; decreased minute ventilation
SO_2	2.6	2 hr	Monkeys	No observed effects
SO_2	2.6-26.0	20-40 min	Dogs	Increased pulmonary resistance; decreased lung distensibility
SO_2 + O_3	0.5-2.1 + 0.4-1.6	2 hr	Guinea pig	No interactive effects from pollutant combination

3.1.3 Clinical Studies

Clinical studies with human subjects provide valuable information on the effects of SO_x exposure. Detailed medical demographic histories of the subjects can provide a sound basis for defining the test group, investigations can be performed under controlled conditions, and an individual can be examined before and after a well-characterized pollutant exposure; therefore, highly accurate determinations of health effects can be made. But in such research, the rights and safety of the subjects are paramount; therefore only short exposure times are possible, the study parameters are limited, and the subjects are often young and healthy. Thus, it is difficult to extrapolate the results of such research to the population as a whole, which varies in terms of age, sex, health status, pollutant exposure experience, and other relevant factors.

In attempting to relate dose-response effects in the laboratory to those occurring under ambient conditions, many parameters must be considered. For instance, it should be noted that a low dose causing some subjective discomfort or after-effect in an individual may not be measurable in the clinical setting. On the other hand, a slight effect measured in the upper airways during a clinical exposure may go unnoticed by the experimental subject. Such an effect would also go unnoticed by an individual exposed under ambient conditions.

Sulfur dioxide at concentrations which may exist during air pollution episodes has been found to exert measurable effects on lung function in the laboratory. Within the concentrations tested, the effects of this pollutant generally are transitory. Most human subjects respond to 5 ppm (13,000 $\mu g/m^3$) SO_2, while more sensitive subjects respond to 1-2 ppm (2600-5200 $\mu g/m^3$) SO_2. The typical response is an increase in pulmonary flow resistance, although at lower levels, e.g., 1 ppm SO_2 (2600 $\mu g/m^3$), significant decreases in nasal mucus flow rate have been demonstrated. Moreover, several investigators have noted transitory effects on pulmonary flow resistance at concentrations slightly below 1 ppm (2600 $\mu g/m^3$) SO_2. A greater increase in specific airway resistance (airway resistance times specific gas volume) has been noted in subjects breathing 1-3 ppm SO_2 by mouth than among those breathing by nose.

Mucus traps microbes and other fine particles inhaled from the ambient air and aids in pulmonary clearance. Thus a reduction in its flow rate by SO_2 could increase susceptibility to infectious diseases. However, when human subjects were inoculated with a virus and exposed to SO_2, the 50 percent decrease in nasal mucus flow rate observed in the subjects was not associated with an increase in the number of colds.

The finding that NaCl potentiates the effects of SO_2 in animals is supported by some but not all studies with human subjects. Similar findings have also been noted for SO_2/O_3 combinations. The most recent of these shows that the combination of SO_2 and O_3 does not affect pulmonary mechanics more than does O_3 alone. This lack of potentiation correlates with recent work on guinea pigs.

Limited data are available from laboratory exposures of human subjects to various sulfate salts and mixtures. Generally, the clinical effects of H_2SO_4 vary with relative humidity, particle size, and individual response. Concentrations of 0.35 to 5.0 mg/m^3 H_2SO_4 have been shown to cause bronchoconstriction and increases in respiration rate, coinciding with decreases in deep breathing. Concentrations of 1 mg/m^3 have been shown to affect mid-expiratory flow rate after short-term exposures. On the other hand, when human subjects were exposed to various sulfates (acid and salts) at concentrations and under conditions simulating an air pollution episode, little or no adverse health effects were observed. In this study 30 percent of the sulfuric acid in the chamber was neutralized, apparently by ammonia exhaled by the subjects. This neutralization reaction seems to mitigate some effects of H_2SO_4 through neutralization. It was suggested that adverse effects from sulfates probably occur through additive and/or synergistic interactions with coexisting pollutants.

The results of clinical studies are summarized in Table 3-3.

3.1.4 Epidemiology

Epidemiological studies show that sulfur oxides affect health and that the degree of the effect is related to the degree of pollution. National air quality standards have been established for sulfur dioxide and for airborne particulate matter, which includes sulfates. However, there is no standard specifically for sulfates, mainly because the public health effects of airborne sulfate compounds have not been delineated.

Table 3-3. EFFECTS OF SULFUR IN MAN

Chemical	Concentration, mg/m^3	Time	Effect	Reference
SO_2	0.96-1.8	20 sec-15 min	Decreased alpha wave in EEG, increased light sensitivity	Bushtueva et al. (1962) Dubrovskaya (1960)
SO_2	0.78-15.6	Up to 120 hr continuous	Changed S-sulfonate plasma levels	Gunnison and Palmes (1974)
SO_2	2.6, 13, 33	10-30 min	Increased pulmonary resistance (dose-response effects)	Frank et al. (1962)
$SO_2 + NO_2$	13 + 10.3	2 hr	Decreased arterial O_2 tension, increased pulmonary resistance	von Nieding et al. (1977)
$SO_2 + H_2SO_4$	0.9 + 0.6	3-15 min	Changed optical response	Bushtueva (1961)
$SO_2 + O_3$	1.05 + 0.72	2 hr	Decreased pulmonary function	Hazucha and Bates (1975)
$SO_2 + O_3$	1.0 + 0.8	2 hr	Decreased pulmonary function attributed to O_3 alone	Horvath and Folinsbee (1977)
SO_2 + NaCl	4.6 + 160	5 min	Increased pulmonary resistance	Toyama (1962)
H_2SO_4	0.35-5.0	5-15 min	Reflex bronchoconstriction, shallow rapid breathing, and decreased minute ventilation	Amdur et al. (1952)
H_2SO_4	0.6-2.4	60-120 min	Transient increased light sensitivity	Bushtueva (1960, 1962)

Toxicological studies show that sulfuric acid and other sulfates affect health only at levels much higher than those normally found in ambient air. Consequently, epidemiological studies are crucial in determining levels that may affect public health. While both mobile and stationary sources emit sulfur dioxide and sulfates (acids and salts), an epidemiological appraisal of the effects of mobile source emissions must take into account the relatively small contribution of this source to the total amount of emission. To date, epidemiological studies on sulfur compounds have focused primarily on stationary sources, which at present are responsible for a nationwide average of about 98 percent of the sulfur emissions.

Early air pollution episodes, such as those occurring in the Meuse Valley in 1930, London in 1952, and Donora, Pennsylvania, in 1948, heightened both public and scientific awareness of the effects of air pollution on public health. During these episodes, extremely high levels of sulfur dioxide and particulate matter (smoke) were related to increases in hospital admissions and deaths. A review of major air pollution episodes that occurred in London and Rotterdam between 1952 and 1962 indicates that excess mortality resulted when 24-hr mean levels of SO_2 exceeded 500 $\mu g/m^3$ for a few days. Moreover, absenteeism and hospital admission for respiratory illness increased whenever 24-hr mean levels of SO_2 reached 300-400 $\mu g/m^3$ for 3-4 consecutive days.

Other epidemiological studies have focused on the health effects of long-term, low-level exposure as opposed to these episodic exposures. For example, study of mortality patterns and pollution levels over a 4-year period in the New York metropolitan region showed that death rates correlated with SO_2 concentrations. Taking into account other variables affecting mortality (such as extreme weather conditions), they determined that mortality was 1.5 percent less than average on days when SO_2 levels were below 0.03 mg/m^3 and 20 percent higher than average on days when SO_2 levels exceeded 0.5 mg/m^3. A long-term study of British school children revealed increased frequency and severity of lower respiratory diseases associated with annual mean smoke concentrations above 130 $\mu g/m^3$ and sulfur dioxide levels above 130 $\mu g/m^3$. Both acute and chronic upper respiratory disease in children have also been epidemiologically linked to sulfur oxide exposure. Another study on 819 school children in Sheffield, England, disclosed that both upper and lower respiratory tract infections were related to actual air pollution measurements. The mean daily averages measured at a single station were 300 $\mu g/m^3$ for smoke and 275 $\mu g/m^3$ for SO_2.

Studies such as these provide the basis for current air quality standards for sulfur dioxide.

In 1974, the U.S. Environmental Protection Agency published epidemiological data relating health effects to sulfur dioxide and sulfates. The data from this report were derived from the Community Health and Environmental Surveillance System (CHESS), which was part of a larger program to assess the impact of air pollution on health. Some interpretations of the CHESS data showed health effects occurring at sulfate levels of 10-15 $\mu g/m^3$. However, these findings have been called into doubt by a Congressional investigation, which identified many problems in the CHESS project. While efforts have been initiated to reevaluate the CHESS data, the precise relationships between sulfates and health effects have yet to be validated.

There are virtually no epidemiological data relating sulfur emissions to health effects on persons living near heavily traveled roadways, on automobile and airplane operators and passengers, or on bikers or pedestrians. Such individuals could be exposed to sulfur pollutants contributed in large measure by mobile sources. Data from a Los Angeles catalyst study provide a good empirical estimate of roadway exposure in one large metropolitan area. On the basis of nearly 2 years of sampling, average 24-hr freeway contributions of sulfur dioxide and suspended sulfate were estimated at about 12 and 2 $\mu g/m^3$, respectively.

In rare instances, ambient sulfate concentrations could amount to several micrograms per cubic meter and be associated with health effects. However, there are no sound epidemiological data to document these effects.

3.2 WELFARE

As seen in the preceding section, sulfur dioxide and sulfates are the primary sulfur-bearing compounds of interest because of their toxicity and abundance. This section examines the effects of these and several other sulfur pollutants on vegetation, nonbiological materials, and visibility and climate.

3.2.1 Vegetation

Sulfur pollutants are harmful to plant life, but the role of mobile source emissions in causing this damage has not been determined. Transportation-related emissions of sulfur dioxide are potentially the most injurious, particularly in combination with other airborne pollutants. Sulfur dioxide was the first pollutant recognized as a phytotoxin, and reports dating back to the 1880's relate this compound to crop injury. After the theory of invisible injury (e.g., slower growth rates) was proposed in 1923, other parameters of sulfur dioxide phytotoxicity were studied. The only positive effects of plant uptake of atmospheric sulfur dioxide occur in areas with low-sulfur soils. In these areas, fumigation or fertilization with sulfur-bearing compounds has been shown to increase the yield of certain crops.

The damaging effects of sulfur compounds on plants are often accompanied by observable symptoms. The symptoms of both acute and chronic exposure to SO_2, including discoloration, partial necrosis, and leaf shedding, have been documented for a number of plants. Less information is available on the effects of exposure to H_2S and H_2SO_4. Plant damage may also occur without apparent signs; for example, it was found that SO_2 reduced photosynthesis in fir and spruce that showed no visible symptoms.

Some particularly sensitive plants have become known as bioindicators for the detection of air pollution. Efforts have been made to develop certain plant species as bioindicators to aid in air pollution monitoring. Studies have established dose-response relationships for injury from SO_2

exposure for various trees, food crops, lichens, and bryophytes. Dose-response injury data are also available for H_2S and COS (carbonyl sulfide), but no definitive data are available for sulfuric acid aerosols.

The sensitivity of plants to the effects of sulfur compounds may be greatly affected by varying environmental conditions, including temperature, light, humidity, carbon dioxide concentrations, soil moisture, and soil nutrients. Moreover, the interaction of SO_2 with other airborne pollutants may alter vegetational injury effects, resulting in less injury, more injury, or synergistic effects, depending upon the pollutants and plant species. Synergistic effects of SO_2 and O_3 have been documented for a variety of plants.

Sulfur absorption rates and resistance have been shown to vary with different plant species exposed to SO_2. For example, sulfur dioxide has been shown to induce stomatal closure in some plants and to induce stomatal opening in others. Tolerance to SO_2, it has also been suggested, may in some instances depend less on the amount of pollutant absorbed than on the ability of the plant to move SO_2 out of the leaf and into other plant tissues.

Recent studies have been made of the effects of SO_2 on microorganisms, including fungi, algae, and bacteria. Growth reductions in these lower organisms have been observed with high-level laboratory exposure, but the direct effects at ambient levels appear minimal. It should be noted, however, that since SO_2 affects soil pH, leaf surfaces, and related factors, the associated microorganisms may also be affected.

Sulfur dioxide is harmful to some plant parasites. Studies have shown, for example, a reduction of mildew infection, fungicidal action, and prevention of mold and rot in certain plants. But SO_2 does not harm all parasites. For instance, it has no effect on tomato blight. SO_2 may or may not repel lice, depending on plant species infested. No direct effects of SO_2 on plant pathogenic bacteria have been reported. More importantly, SO_2 generally weakens plants and thereby renders them more vulnerable to parasitic attack.

Atmospheric loading of SO_x and NO_x as emitted from fossil fuel combustion seems to be responsible for the increasing acidity of rain in many geographic areas. The available literature indicates that pollution in rainfall may cause direct injury to foliage, deposition of other harmful substances on the plant tissue, effects on host-parasite relationships, and effects on soil fertility. Studies have shown, for example, that the acidic nature of SO_2 at high humidity (sulfurous acid) may degrade chlorophyll and plasmolyze plant cells. Acidic rainfall has also been linked to reduced plant growth, although in some areas it may actually increase the nutrient supply of nitrate and sulfate ions, common components of chemical fertilizers.

3.2.2 Nonbiological Materials

The damaging effects of air pollution on nonliving objects can be quantified in economic terms by a materials damage estimate. This estimate, based on complex methods of data selection and analysis, helps to assess the relative costs and benefits of attempts to reduce or prevent pollution.

However, not all materials damage can be expressed in economic terms. In many parts of the world, air polluted with sulfur oxide is silently eating away works of art. Some of the finest monuments of antiquity and thousands of pieces of sculpture and carvings on historic buildings such as cathedrals show vivid evidence of the insidious effects of polluted atmospheres. While no economic cost can be applied to this damage, the loss to society is permanent.

Mobile sources account nationwide for an average of less than 2 percent of all manmade sulfur compound emissions (stationary sources account for most sulfur emissions). There are no data relating materials damage to specific levels of certain sulfur-bearing compounds. Since it is not possible in ambient air measurements to identify unequivocally the source of these compounds, the only available way to estimate damage by a sulfur-bearing compound from mobile sources is to multiply the overall damage values by the relative proportion of mobile sources to all sources (2 percent). No studies have been conducted to isolate the relative contribution of mobile versus nonmobile sources in particular areas, such as roadways, where the mobile source contribution might be much higher than the nonmobile source contribution.

In terms of potential damage to materials, the most significant sulfur compounds emitted from mobile sources are sulfur dioxide (SO_2), hydrogen sulfide (H_2S), sulfuric acid (H_2SO_4), and absorbed sulfates ($SO_4^=$). Less significant species such as organic sulfides, disulfides, and thiophenes are present, if at all, at relatively low concentrations.

Since these organic compounds react minimally with common nonliving materials, they are not considered in this report.

It is important to consider damage to materials by the type of sulfur compound emitted by mobile sources. The various species of sulfur compounds emitted directly from these sources are often transformed into other species. These transformed products may have damaging effects different from those of the original compounds. Therefore, knowing what compounds are emitted is not always sufficient to predict subsequent damage to materials.

Generally, air pollutants damage materials by five principal mechanisms: abrasion, direct and indirect chemical attack, corrosion, and particle deposition and removal. The damaging action of pollutants can also be enhanced synergistically through interaction with natural factors such as moisture, particles, temperature, sunlight, air movement, physical position, and biological action.

Thousands of materials are exposed to sulfur emissions from mobile sources. Since a materials damage study cannot account for all damage to all materials, a scheme is needed to identify those materials whose damage has the greatest economic impact. In general, to warrant detailed study a material should both be affected by the pollutant of interest and be economically important.

A comprehensive recent review of the economic effect of air pollutants was assembled from several studies that used both comparative and analytic approaches and covered various aspects of the problem. The review includes studies of general materials damage, damage to electrical contacts corrosion to metallic systems and structures exposed to the outdoor atmosphere, damage to electrical components, and deterioration of exterior paints.

3.2.3 Visibility and Climate

High atmospheric concentrations of sulfates reduce visibility, and the possibility that they play a role in climatic effects has also been considered. Mobile sources produce sulfates directly as well as indirectly, in that the sulfur dioxide they emit can be oxidized to sulfates in the atmosphere. However, these sources account for only a small portion of the total atmospheric sulfates, so small in fact that they would not in themselves measurably reduce visibility or exert any effects on climate. The following discussion defines relationships between sulfur-bearing compounds and visibility reduction and also addresses the question of climatic effects. Visibility reduction will be explored in greater detail in a separate, later report required under Section 169(a) of the 1977 Amendments to the Clean Air Act.

Recent studies suggest that sulfates may be one of the primary causes of visibility reduction associated with air pollution in some areas. For example, regression models based on daily variations in visibility and pollutant concentrations indicate that sulfates may account for half of the total visibility reduction in the east. Widespread atmospheric hazes throughout the east are increasing with regional SO_x emissions.

There is concern over transport of sulfates into relatively unpolluted areas from long distances. Impairment in visibility in these areas could occur by transport of urban and other industrial pollutants, and these effects may equal or exceed effects from closer pollutant emissions. Highlighting this concern, a recent review showed that sulfur transported from the United Kingdom arrived in Scandinavia as sulfates. Another study noted the effects on regional visibility of a 9-month strike in the copper-smelting industry in Arizona. During the strike, ambient sulfate levels dropped significantly at distances up to 500 km from the source areas, and visibility improved 5 to 25 percent at locations within 250 km of the smelters.

Sulfur-bearing compounds known to reduce visibility are ammonium sulfate, sulfuric acid mist, and ammonium acid sulfate. A Los Angeles study relating aerosol composition to light-scattering measurements indicated that sulfates and nitrates always dominate the scattering but that one may be more important than the other, depending on the location of measurement. Whether these relationships hold in other geographic areas, at different pollution concentrations, and with different meteorological conditions remains to be seen. However, most available studies indicate that sulfates are a primary cause of visibility reduction. The added contribution from nitrates seems to vary according to relative humidity.

The results of an EPA-sponsored smog chamber study demonstrated the contribution of a number of factors to the visibility-reducing effects of automobile exhaust. While the ratio of HC/NO emissions was important, it was shown that visibility reduction was also correlated with SO_2 and relative humidity (RH). Under the controlled conditions of the test, the effects of these two factors, which were interactive, increased with irradiation time. When RH was low (35 percent), the exhaust emission's effect on visibility changed little over the course of the irradiation period. At the other extreme, when the RH was raised to 80 percent and the air-exhaust mixture was supplemented with additional SO_2 (0.9 ppm), visibility fell from an initial value of about 50 km to less than 4 km within 22 hr. Analysis of data from tests of different fuels indicated that the sulfur content of gasoline was directly related to emissions which led to the formation of light-scattering aerosols. Thus, at a given ratio of HC/NO emissions, fuels with high sulfur content had the most marked effects on visibility in this chamber study.

In addition to reducing visibility, pollutants released to the atmosphere may alter the environment by causing slow and subtle changes in atmospheric composition and, possibly, climate. The release of gases and production of aerosols could significantly alter the atmosphere's composition (hence, heat absorption characteristics), bringing about a change in the earth's radiation balance and a concomitant temperature change. Cloud formation might also be affected, since changes in cloud cover could alter albedo (reflection of sunlight), another important factor in maintaining the earth's radiation balance. Changes in the location, frequency, and amount of precipitation might also occur.

So far as is known, only sulfur compounds in solid phase (i.e., sulfates) may be involved in climatic processes. In fact, for all practical purposes, this discussion can be limited to sulfate particles. These particles absorb sunlight and water from the atmosphere, and they can also affect cloud condensation nuclei (CCN), which are critical in the rainfall process.

Many reports have correlated particulate matter with potential climate change. Natural events such as the explosive injection of volcanic material into the atmosphere verify this correlation by showing the capacity of volcanic material to scatter incoming solar radiation. For example, an analysis of upper air temperatures in the Southern Hemisphere following the Mt. Agung eruption in 1963 detected a major warming attributed to the absorption of radiation by the aerosol. Few surface temperature or rainfall consequences have been linked directly with volcanic events, but it should be noted that normal variability of weather would obscure all but the most dramatic effects.

On a global scale, meteorological observations linking air pollution to climate changes are lacking. Over cities, certain effects on weather have been identified. For example, the reduction of received radiation in some urban areas can be ascribed definitely to the increase of fine aerosols. Cloudiness appears to be increased by 5 to 10 percent in these areas. Generally speaking, fog develops about twice as often over cities as over the countryside, and the duration is lengthened by an abundance of hygroscopic compounds. Thunderstorms may be enhanced, and precipitation appears to be increased over and especially downwind of cities. However, the effects of pollutants on precipitation have yet to be separated from the effects of thermal energy over urban areas. Moreover, studies of air pollution and local or regional climatic processes have not yet defined a role for mobile source emissions.

4. CONTROL ALTERNATIVES

4.1 SUMMARY

This chapter reviews various alternatives that have been investigated to control vehicular emissions of sulfur-bearing compounds. Low-emissions technology and on-board control devices have been explored primarily with regard to emissions from catalyst-equipped, gasoline-powered vehicles. Two additional areas of study are also considered--desulfurization of fuel and short-term allocation of low-sulfur crude oils--which have relevance to all mobile source emitters.

The focus is on sulfates, which, potentially, are the most hazardous sulfur-bearing pollutants emitted by mobile sources. No data on control alternatives are available for other sulfur compounds, such as H_2S and COS, which are emitted infrequently and at low levels under normal operating conditions.

In the field of low-emissions technology, controlling oxygen levels through air injection is considered one of the more promising alternatives for sulfate reduction. At present, air injection systems used with catalysts designed to meet HC and CO emissions standards introduce additional oxygen that increases sulfate emissions. However, because HC and CO are more readily oxidized than SO_2, sulfate production from the oxidation of SO_2 may be reduced by carefully limiting the amount of air made available to the catalyst.

Several catalyst formulations with various materials and design configurations have been investigated. While this approach may eventually yield viable results, no practical alternatives have yet been demonstrated. Likewise, studies of elevated catalyst temperature to enhance HC, CO, and NO_x combustion efficiency and at the same time reduce sulfate production have not yet achieved low-emissions results.

On-board control devices--sulfate traps--have been the subject of several studies. Traps tested thus far have demonstrated problems of efficiency and durability, and considerably more research would be needed to achieve practical results from this technology.

Desulfurization of fuel during the refining process is another possible control alternative. Reasonably obtainable target reductions in the sulfur content of gasoline, diesel fuel, and jet fuel are examined in terms of the costs involved to meet production demands through 1990.

Finally, the short-term allocation of low-sulfur crude oils is examined as a temporary measure to reduce sulfate emissions while other control alternatives are being implemented.

4.2 LOW-EMISSIONS TECHNOLOGY

4.2.1 Catalyst Inlet Oxygen Level Control

Work done by EPA, Exxon, GM, Chrysler, Volkswagen, and Engelhard has shown that sulfuric acid emissions can be reduced by lowering the amount of oxygen available to the catalyst (Bachman et al. 1976; U.S. Environmental Protection Agency 1976b, 1977). In control systems not incorporating air pumps, the exhaust fed to the catalyst has an oxygen content of up to 5 percent, but usually ranges between 2 percent and extremely low levels approaching zero. This range is directly related to engine calibration. Thus, calibration on the lean side of stoichiometric proportions leads to higher exhaust oxygen levels, while rich calibration leads to lower levels.

In converter systems equipped with air pumps, the exhaust fed to the catalyst has an oxygen content ranging up to 7 percent, depending on the rate of air injection and engine calibration (U.S. Environmental Protection Agency 1976b, 1977; Somers et al. 1977). Sulfate emissions from vehicles so equipped could be reduced to about the level of those from non-catalyst vehicles by limiting the amount of excess oxygen reaching the catalyst (see Table 4-1). However, HC and CO control may suffer at the same time.

Chrysler investigations have indicated that catalyst oxygen levels at or below 1 percent are sufficient to greatly reduce sulfate emission (Figure 4-1) (U.S. Environmental Protection Agency 1976b). A similar finding has been made by Nissan (U.S. Environmental Protection Agency 1977). This study shows that an air/fuel ratio (an indicator of the amount of oxygen present) of 14.5 or less is related to low sulfate emissions, while a sharp increase in sulfates occurs between ratios of 14.5 and 15.5.

Table 4-1. O_2 LEVEL EFFECTS

							Catalyst										
Tested by:	Model	CID	AIR	EGR	P	M	Type	CID	Active materials	T exh, °F	Excess O_2 level, %	HC, gpm	CO, gpm	NO_x, gpm	H_2SO_4, mgpm	H_2SO_4 precondi- tioning	H_2SO_4 test cycle
Exxon	'75 Chevrolet	350	Warm-up		AC		Ox			870		0.17	2.38		2		FTP #1
										1200	0.8				53		60 SS-1 (1/2 hr)
										1200					20		60 SS-2 (1/2 hr)
										1200					15		60 SS-3 (1/2 hr)
										1200					15		60 SS-4 (1/2 hr)
										890		0.21	3.44		0.6		FTP #2
			Full				Ox			870		0.19	2.28		10		FTP
										1150	3.5				98		60 SS - 1
										1150					58		60 SS - 2
										1150					44		60 SS - 3
										1150					36		60 SS - 4
												0.12	2.26		3		FTP
Exxon		351	Warm-up			x	Ox					0.22	4.90		3		FTP
															5		60 SS - 1
															2		60 SS - 2
															3		60 SS - 3
															4		60 SS - 4
												0.33	5.73		3		FTP
			Full			x	Ox					0.19	2.16		20		FTP
															56		60 SS - 1
															44		60 SS - 2
															44		60 SS - 3
															44		60 SS - 4
												0.19	2.16		23		FTP

Table 4-1 (Continued). O_2 LEVEL EFFECTS

							Catalyst					'75 FTP					
Tested by:	Model	CID	AIR	EGR	P	M	Type	CID	Active materials	Mileage	Excess O_2 level, %	HC, gpm	CO, gpm	NO_x, gpm	H_2SO_4, mgpm	H_2SO_4 preconditioning	H_2SO_4 test cycle
Engelhard	'72 Galaxie	351	Full	x		x	Ox	153			4-7[a]				82	AMA	SC-7
						x	Ox	153			4-7[a]				18	AMA	SC-7
						x	Ox	153			4-7[a]				54	AMA	SC-7
Engelhard	'75 Torino	351	None	x		x	Ox	153	Pt/Pd	25,000	0.3-1.5[a]	0.37	4.05	1.66	4.2	9 mi of city and highway driving	75 FTP
EPA	Cutlass		None		AC		Ox			2,500	4[b]	0.41	1.59	1.23	2.7	1000 mi of AMA	75 FTP
															16.8	Above	SC-7
															16.6	Above	SC-7
															34.5	Above	HFET
															21.4	Above	SC-7
												0.48	1.57	1.25	1.1	Above	75 FTP
															37.3		SC-7
															39.1		SC-7
															53.4		HFET
															41.9		SC-7
															29.5		SC-7
EPA	Cutlass		None		AC		Ox			7,000	1[b]	0.37	3.76	0.91	2.4		75 FTP
															2.9		SC-7
															2.1		SC-7
															4.5		HFET
															2.4		SC-7
															2.6		SC-7
												0.55	4.29	1.05	1.7		75 FTP
															1.9		SC-7
															2.0		SC-7
															3.4		HFET
															2.8		SC-7
															2.8		SC-7
															2.8		SC-7
															3.9		SC-7
															2.7		SC-7
															3.1		SC-7

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[a] Total O_2 level out of catalyst.

[b] Total O_2 level to catalyst.

Source: U.S. Environmental Protection Agency (1976b).

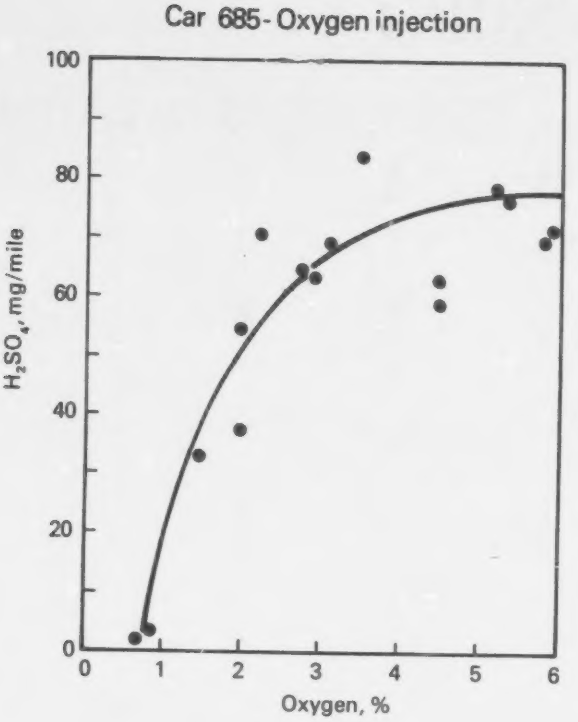


Figure 4-1. Sulfuric acid emissions as a fraction of oxygen injection level. 318 Chrysler with air pump and monolith catalyst.

Source: U.S. Environmental Protection Agency (1976b).

As mentioned above, reductions of the oxygen level in oxidation catalyst systems may result in an increase in HC and CO emissions. Future advances in oxidation catalyst technology may overcome this problem. Three-way catalyst systems, which have an inherently low oxygen level, tend to have low sulfate emissions without impaired control of HC and CO emissions.

4.2.1.1 Modification of air injection with oxidation catalyst systems--Current oxidation catalyst-equipped cars make use of an engine-driven pump which introduces air into the exhaust system upstream of the catalyst. There are several potential methods by which the function of this pump can be controlled to limit the oxygen content of the exhaust stream.

4.2.1.1.1 Diverter valve. Exxon Research and Engineering used a simple diverter valve to inject air into the catalyst during the warmup part of the cold start (Bachman et al. 1976). This system reduced sulfate emissions, but it also reduced the control of CO emissions. Loss of CO control was worse with the monolith catalyst systems tested than with the pelleted catalyst systems, possibly because there was less surface area and active metal present in the former.

4.2.1.1.2 Air modulation. Recent EPA work under contract with Southwest Research Institute indicates that a more sophisticated air modulation device may make it possible to maintain HC and CO emission control while reducing sulfate emissions (Ingalls and Springer 1976). This device uses engine vacuum input to regulate the air injection system. Enough oxygen is injected to oxidize the spikes of HC and CO that occur during a typical driving cycle, but the overall excess of oxygen is not so high as to cause sulfate production. Preliminary results indicate the possibility of attaining mandated emissions levels of HC, CO, and NO_x while minimizing sulfate emissions (Ingalls and Springer 1976). The durability of this system is currently being tested. Another system that may benefit from the use of a modulated air injection system is the combination of a three-way catalyst and an oxidation catalyst used by Ford for 1978 in California and being considered by some other auto manufacturers. Ford has indicated that this system is their prime candidate for meeting statutory emission standards of 0.41, 3.4, and 0.4 g/mile HC, CO, and NO_x (U.S. Environmental Protection Agency 1977). The current high sulfate emissions from this system (see Table 4-2) are caused by the high oxygen level associated with full air injection between the three-way and the oxidation catalysts. However, the use of modulated air injection on such systems may reduce high sulfate emissions without loss of HC and CO control. The auto manufacturers are investigating this possibility.

4.2.1.1.3 Three-way catalyst systems. The most promising control system for use in cars designed to meet 1980 emissions standards is the three-way system, which consists of two major components: (1) a catalyst which has both oxidation activity (catalyzes the conversion of CO and HC to CO_2 and water) and reduction activity (NO_x to N_2) and (2) a continuous fuel/air ratio monitoring and control device which maintains both adequate oxygen and reductant levels in the exhaust. Several vehicles manufactured by GM, Ford, Volvo, Saab, and Mercedes now employ this type of control system (U.S. Environmental Protection Agency 1977).

Several studies have shown that three-way control systems normally produce very low emission rates of sulfuric acid (Tables 4-1, 4-3, and 4-4). Two factors appear to be responsible for these low rates: low oxygen content of the exhaust and inherently low sulfate-forming tendencies of the Pt/Rh catalyst compositions needed for NO_x control.

Table 4-2. SULFATE EMISSIONS FROM THREE-WAY PLUS OXIDATION CATALYST PROTOTYPES WITH AIR INJECTION

System	HC, g/mi	CO, g/mi	NOx, g/mi	H_2SO_4 mg/mi[a]
8D1-302-7P (3-way and oxidation catalyst)	0.14	0.40	0.49	27.9
	-	-	-	33.3
	0.13	0.43	0.50	25.8
	0.14	0.29	0.49	34.5
	0.13	0.28	0.61	37.4
	-	-	-	43.0
	0.13	0.20	0.67	45.7
	0.14	0.26	0.64	40.1
8D1-302-1P (3-way and oxidation catalyst)	0.15	0.16	0.60	56.2
	-	-	-	51.2
	0.16	0.14	0.70	61.4
	0.15	0.13	0.67	62.3
	0.16	0.17	0.55	69.8
	-	-	-	53.6
	0.18	0.14	0.55	71.4
	0.18	0.18	0.56	61.2

[a] All data over CUE driving schedule.

Source: U.S. Environmental Protection Agency (1977).

Table 4-3. 3-WAY CATALYSTS

							Catalyst					'75 FTP					
Tested by:	Model	CID	AIR	EGR	P	M	Type	CID	Active materials	Mile-age	Excess O_2 level, %	HC, gpm	CO, gpm	NO_x, gpm	H_2SO_4, mgpm	H_2SO_4 precondi-tioning	H_2SO_4 test cycle and comments
Exxon	Volvo[a]					EI	Ox				0.9-1.4	0.17	1.37	2.24	1.1		FTP, lean closed loop FI
						EI	3-Way			4,000	0	0.14	1.04	0.56	0.8		FTP, closed loop FI
										16,000	0	0.23	2.12	0.46	1.7		FTP, HWFET
															0.4		
										0		0.25	2.00	0.41			
Toyota		1600 cc	None	x	Toy		3-Way		Pt/Rh	20,000		0.43	2.56	1.47	0.2	30 mi at 30 SS	Not 72 FTP, closed loop FI
															0.08		50 SS
															0.1		30 SS
															0.08		50 SS
															0.1		Hot 72 FTP
EPA	Volvo	128	None	None		EI	3-Way	102	TWC-1	4,000	0.2[b]	0.219	0.735	1.73	0		75 FTP
															0		HWFET
															0		60 SS
Exxon	Volvo	128	None	None		EI	3-Way	102	TWC-9	4,000		0.22	1.93	0.87	6.9	16.6 9 mi of city & highway driving	75 FTP, closed loop FI
										4,000		0.21	1.40	0.91	1.2	16.1	75 FTP
										14,000					2.1		75 FTP
															1.2	FTP	55 SS
															0.4	Above + 45 mi of 55 SS	55 SS
															0.3	Above + 55 mi of 55 SS	30 SS
															0.4	Above + 30 SS	HWFET
VW	Beetle		None			x	3-Way		Pt/RH			0.18	3.27	0.62	2		75 FTP, closed loop FI
EPA	Volvo		None							15,800		0.13	1.42	1.66	2.6	500 mi of AMA	75 FTP closed loop FI
															1.6		SC-7
															1.7		SC-7
															2.0		HFET
															1.3		SC-7
															1.4		SC-7

[a] Lean biased fuel injections.

[b] Total oxygen level.

Source: U.S. Environmental Protection Agency (1976b).

Table 4-4. SULFURIC ACID EMISSIONS OF THREE-WAY CATALYST PROTOTYPES

Car	Control system	Fuel sulfur level, %	Exhaust oxygen level, %	Sulfuric acid, mg/mi
'74 Chevrolet	Closed-loop fuel injection, 3-way catalyst	0.06	0.7	0.2
'74 Chevrolet	Closed-loop fuel injection, 3-way catalyst	0.06	0.3	0.1
'74 Chevrolet	Closed-loop carburetor, 3-way catalyst	0.031	0.7	0.2

Source: U.S. Environmental Protection Agency (1977).

However, a variation on the three-way emission control system (Section 4.2.1.1.2), currently produced by Ford and considered by other manufacturers, has been shown to emit high levels of sulfates. This system combines a three-way catalyst (TWC) and an oxidation catalyst with air injection together with an oxygen sensor feedback controlled carburetor. The added oxidation catalyst is needed with this system to provide the additional CO control needed to meet the 3.4 g/mile CO standard, according to a Ford spokesman. However, EPA feels that there are alternate ways of meeting the 3.4 g/mile CO standard with the three-way systems without excessively high sulfate emissions. Substitution of a closed-loop fuel injection system for the carburetor is one potential way to meet the 3.4 g/mile CO standard as evidenced by Volvo and Saab three-way vehicle devices (Griffith et al. 1978; U.S. Environmental Protection Agency 1977). A fuel injection system is superior to a carburetion system in maintaining a precise stoichiometric control and distribution of air/fuel ratio. This precise control leads to better CO control in the TWC and hence eliminates the need for an added oxidation catalyst. However, there are substantial price differences between fuel injection and carburetion. Another method, and possibly the best, of potentially attaining the 3.4 g/mile CO standard with low sulfate emissions with a TWC is to control the air injection with a modulation device similar to that discussed in Section 4.2.1.1.2.

Since three-way catalytic control systems are already being adopted because of NO_x control, and since very low sulfuric acid emissions are normal consequences of their function, this sulfate control alternative has no incremental cost. To put this issue into another context, the sulfuric acid control obtained by three-way systems costs nothing because the system is already needed for statutory control of NO_x. However, use of a three-way plus oxidation catalyst that is also a low sulfate emitter may require an air injection modulation system, which would have some costs associated with it.

4.2.2 Catalyst Design

Several studies have indicated that some degree of sulfuric acid control can be obtained by modifying catalyst composition/construction details or operating parameters. For example, it has been found that both base metal catalysts and high-rhodium noble metal compositions have lower H_2SO_4 emissions than do more conventional Pt/Pd mixtures. However, both types suffer from poor durability (U.S. Environmental Protection Agency 1976b).

Perovskite-like minerals as catalyst supports provide a wider range of metal dispersion characteristics than are available with more conventionally used alumina supports. Chrysler and EPA have studied the possibility of modifying metal dispersion and, hence, limiting sulfuric acid formation, but without much success (Bachman et al. 1976). Table 4-5 compares sulfate emissions between GM catalysts and perovskite catalysts.

Theoretically, reducing the time available for SO_2 conversion should reduce the extent of this rate-dependent process. Therefore, reducing catalyst volume or increasing exhaust gas velocity, both of which reduce the residence time of exhaust in the catalyst, should reduce sulfuric acid formation. Only a limited amount of research (by Exxon) has gone into the investigation of this possibility, which has not yet been substantiated or disproven.

Table 4-5. COMPARATIVE SULFATE EMISSION RATES OF THE GM AND PEROVSKITE CATALYSTS IN STANDARD AND HIGH-TEMPERATURE CONFIGURATIONS

GM catalyst

Test	Standard configuration SO_4=, mg/km	Standard configuration Conversion, %	High-temperature configuration SO_4=, mg/km	High-temperature configuration Conversion, %
FTP	6.7	4.9	4.6	3.4
SET[a]	5.6	5.8	1.8	1.9
HET	6.8	8.0	1.6	1.9
64 km/h cruise	8.4	10.6	1.9	2.5

Perovskite catalyst

Test	Standard configuration SO_4=, mg/km	Standard configuration Conversion, %	High-temperature configuration SO_4=, mg/km	High-temperature configuration Conversion, %
FTP	6.6	4.8	3.1	2.2
SET[a]	17.2	18.0	7.4	7.6
HET	21.7	25.0	8.5	9.7
64 km/h cruise	17.8	22.4	8.8	12.2

[a] Average of four tests.

Source: Bachman et al. (1976).

Finally, it should be possible to reduce the thermodynamic equilibrium of H_2SO_4 yield from catalysts by increasing exhaust temperature. Exxon and EPA have investigated this possibility by using insulation, ceramic port liners, and relocation of exhaust elements to minimize heat loss and maximize catalyst temperature. However, over the narrow temperature range available from these changes, no substantial reductions in sulfuric acid emissions were found (Griffith et al. 1978).

In summary, the potential for sulfate control (while regulating HC and CO emissions) by modifying the catalyst type, preparation, or operating conditions appears limited. On the other hand, the three-way system as a control option is very effective, will be needed for NO_x control anyway, and involves no incremental cost.

4.3 ON-BOARD CONTROL DEVICES

4.3.1 Sulfates

While most studies of sulfate emissions have dealt with the engine/emission control system, some research has examined on-board control devices, i.e., sulfate traps. Sulfate traps can generally be divided into two categories: particulate traps and sorbent or chemical traps.

4.3.1.1 Particulate traps--Particulate traps, which use physical means to separate particles from the exhaust stream, have not been considered an effective control alternative for sulfuric acid emissions. Sulfate particulates from mobile sources are generally considered too small to be effectively removed by such physical means. Additionally, in the part of the exhaust system where a particulate trap would be used, much of the sulfate exists in the vapor phase because of the high temperatures there.

4.3.1.2 Sorbent traps--These traps generally use a chemical reaction (i.e., an acid-base type of reaction) to remove sulfate from exhaust. The best candidates for sorbent trap materials are those which are readily incorporated into a trap-effective form and which react with sulfate in the vapor or liquid phase. Extensive work in this area has been done both by Exxon Research and Engineering (under contract with EPA) and by General Motors (Bachman et al. 1976; U.S. Environmental Protection Agency 1976b).

The EPA-funded work at Exxon Research and Engineering included a literature search to identify the most promising sorbents, construction of a laboratory reactor to rapidly evaluate these sorbent candidates, and operation of vehicle tests to demonstrate the actual effectiveness of the materials and designs used. The literature search identified calcium oxide, magnesium oxide, and magnesium and aluminum metals as the most likely sorbent candidates. Many different formulations of these and other compounds were evaluated in the laboratory reactor. Results from these experiments identified the most promising sorbent candidates for later vehicle evaluation.

High sulfate collection efficiency ratings were achieved in the vehicle tests. However, a number of problems were encountered and not satisfactorily resolved. One of the most significant problems was constructing sorbent pellets that were both durable and efficient. These attributes were often in conflict: thus, a pellet of proper hardness (i.e., for durability) tended to be less efficient for sulfate collection than the softer sorbent pellets, which tended to degrade under test and plug the trap, causing a high pressure drop. Another problem was to obtain pellet materials that maintained a high level of sulfate collection without a fall in trap pressure, an effect attributed to the geometry of the pellets used.

GM, relying on the preliminary Exxon literature search and vehicle test data, tried a large number of supported and bulk sorbents, mostly without success (U.S. Environmental Protection Agency 1976b, 1977). The supported sorbent candidates tested (Table 4-6) had not been thoroughly investigated in the Exxon study. These materials were found to be durable, but they showed very low activity as sulfate traps. Further investigations by GM involved testing 40 bulk materials similar to those tested in the Exxon study. A schematic of the test cell used is shown in Figure 4-2. In all this work, GM found only two potential candidates (both bulk sorbents), CaO and Na_2O/Al_2O_3. Vehicle testing of these sorbents showed good initial activity followed by a rapid dropoff in efficiency.

The results of the GM and Exxon investigations indicate that although it may be possible to develop a good sulfate trap, an extensive amount of development work would be necessary before this technology could be ready for production use.

4.4 FUEL DESULFURIZATION

Fuel desulfurization is another alternative to control sulfur emissions. Both sulfur dioxide and sulfate emissions from catalyst-equipped cars and from diesel passenger cars and trucks correlate directly with the amount of sulfur in the fuel. Thus, a 90 percent reduction in fuel sulfur would result in a 90 percent reduction in sulfur emissions from these vehicles. Reducing gasoline sulfur content by 90 percent has been judged prohibitively expensive, but a 65 percent reduction appears practicable (U.S. Environmental Protection Agency 1976a). For diesel and jet fuels, which have a higher sulfur content

Table 4-6. SUPPORTED SULFATE SORBENTS

Noble metals on Al_2O_3	Metal oxides on Al_2O_3	Alkali/alkaline earth oxides on Al_2O_3	Miscellaneous
Pt (6)[a]	Ce	Ba	Ni/SiO_2
Pd (4)	Co	Ca (3)	Cu/ZrO_2
Pt-Pd (3)	Cu (3)	K (3)	Ca/SiO_2
	Cr	Li	Na/SiO_2
	Fe (3)	Na (5)	Ca/MgO
	Mg (2)		Na/MgO
	Mn (2)		Ca/ZrO_2
	Nd		Na/ZrO_2
	Ni		Ca/TiO_2 Al_2O_3
	Pb		Na/TiO_2 Al_2O_3
	Th		
	V		
	Zn (3)		
	Zr		
	Co-Mo		
	Cu-Cr		
	Cu-Mn		

[a] Number in parentheses indicates number of samples evaluated, with different support/active material loading combinations.

Source: U.S. Environmental Protection Agency (1976b).

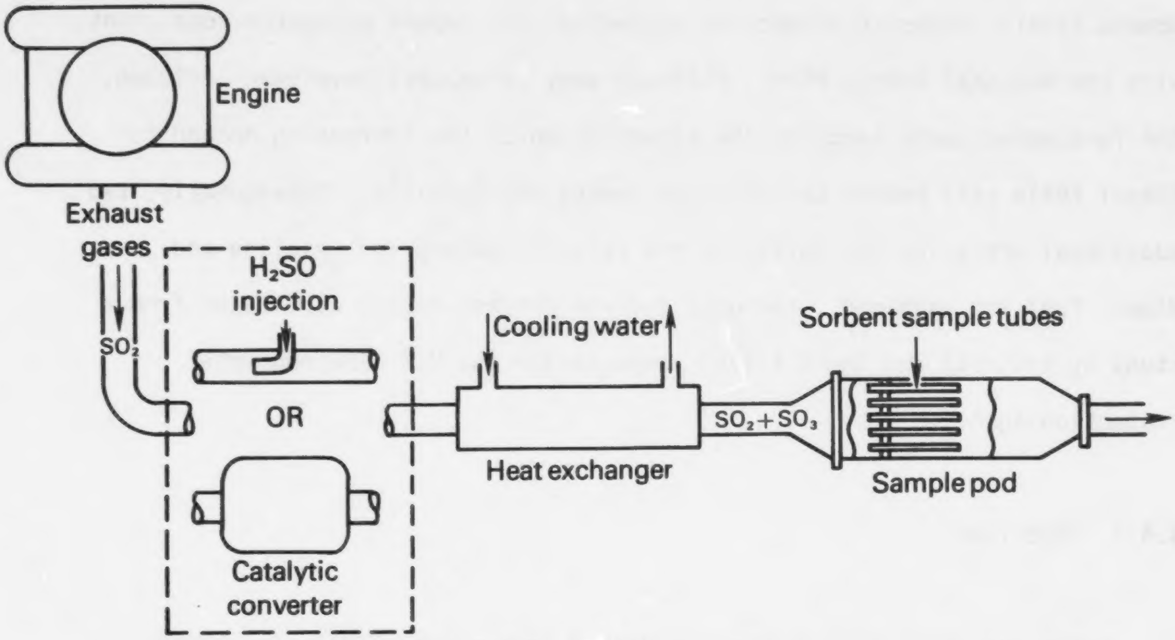

Engine
Exhaust gases
SO_2
H_2SO injection
OR
Catalytic converter
Cooling water
Heat exchanger
$SO_2 + SO_3$
Sorbent sample tubes
Sample pod

Figure 4-2. Test cell evaluation of sulfate sorbents.

Source: U.S. Environmental Protection Agency (1976b).

to begin with, a 90 percent reduction appears economically feasible. Accordingly, an examination is made of the costs to the United States petroleum-refining industry for reducing the average sulfur content of gasolines to 100 ppm, diesel fuels to 200 ppm, and jet fuels to 200 ppm. This report is the first to examine mobile source fuel desulfurization costs with revised product demand levels, domestic production estimates, and import estimates consistent with the National Energy Plan. Although many parameters have been included, the forecasted costs hinge on the extent to which the increasing demand for diesel fuels will reduce the relative demand for gasoline. Consequently, two additional scenarios for shifts in the relative demand for gasoline and diesel fuel are examined. The data for the present report were taken from a study by Kittrell and Short (1978) prepared for the U.S. Environmental Protection Agency.

4.4.1 Gasolines

Motor gasolines produced in the United States vary widely in sulfur content. The Northeast produces gasoline containing roughly twice the sulfur found normally in that produced in the Pacific Northwest (Table 4-7). This regional difference arises from the type of crude oil processed, the processing configuration, and the grades of gasolines produced. Unleaded and premium gasolines contain less sulfur than the regular grade. Nationwide, the average of all grades of gasolines currently ranges between 350 and 400 ppm sulfur; the higher limit occurs during the summer peak production period. In terms of the future, as the demand for unleaded fuel increases, so will the production of low sulfur-containing gasolines. However, there will also be increased

Table 4-7. CURRENT SULFUR CONTENT OF U.S. MOTOR GASOLINES

Representative region	Sulfur content by grade, ppm Unleaded	Regular	Premium
Northeast	315	460	290
South Plains	295	400	320
Pacific Northwest	70	225	115
Southern California	310	600	355
U.S. average	280	415	275

Source: Shelton (1977).

use of crude oils with higher sulfur content. With these offsetting trends taken into account, it is estimated that gasolines produced in the future will have an average sulfur content of about 350 ppm.

The use of diesel and No. 2 fuel oil will increase considerably through 1990 relative to other refinery products. At present, the sulfur content of diesel fuel ranges between less than 200 ppm and 10,000 ppm, with an average of 2000 ppm (Figure 4-3). City bus-type vehicles use fuel with a sulfur content of 1000 ppm, while railroad diesel fuel has a sulfur content of 2500 ppm. Truck and tractor fuels generally contain about 2000 ppm sulfur, but some truck-tractor fuels may contain as much as 10,000 ppm. To meet a 200-ppm target level, more than 90 percent of the diesel fuels must be hydrodesulfurized. Without further sulfur regulations, future sulfur levels will probably rise above 2000 ppm but not exceed 3000 ppm.

4.4.2 Jet Fuels

Approximately 30 percent of the jet fuels marketed in the United States contain less than 200 ppm sulfur, meeting the target specifications of this study. At present, in terms of nationwide averages, commercial jet fuel contains about 600 ppm sulfur, and military jet fuel contains about 420 ppm sulfur. However, military fuels are expected to shift to the higher sulfur content material now used for commercial fuels. Jet fuels now contain about 30 percent more sulfur than those produced before 1973. Without further regulations, this increase will continue, but it should level off at a nationwide average of about 1000 ppm sulfur. Some current jet fuels contain up to 2000 ppm sulfur, and airline specifications allow a maximum of 3000 ppm.

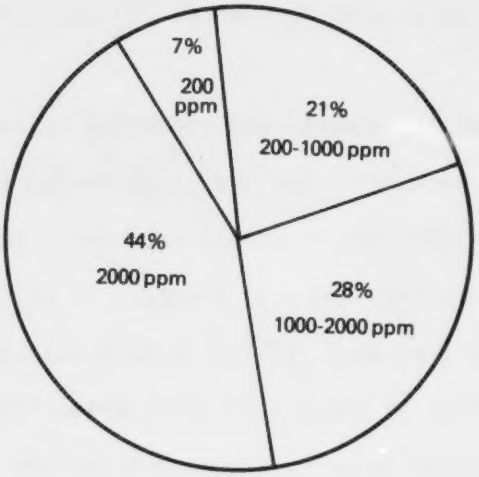


Figure 4-3. Sulfur distribution of U.S. diesel fuels, 1977.

Source: U.S. DOE Diesel Oil Survey (1977).

4.4.3 Economic Impacts

A forecast of the oil needs of the United States through 1990 predicts a peak gasoline demand between 1980 and 1985, followed by a decline--the first such long-term decline in the history of the United States (Table 4-8). According to this forecast, 43 percent of the crude oil processed would be imported. Most of the gasoline processed by crude oil refining [fluid catalytic cracking (FCC) units and coking units] would require desulfurization to attain a 100-ppm sulfur level. Over 70 percent of all jet fuels and over 90 percent of all diesel fuels would require desulfurization to meet the target 200-ppm level.

Estimated capital investments and operating costs for fuel desulfurization are noted in Table 4-9. Because of the lead time needed to install manufacturing facilities, only 1985 and 1990 costs are reported. Capital investments are projected at $6 billion in 1985 and will increase to $6.6 billion by 1990. Manufacturing costs range from $3.3 billion to $3.6 billion for the same dates. Approximately two-thirds of these costs will arise from gasoline desulfurization. The 1990 per-gallon costs noted in Figure 4-4 include a 21 percent energy-manufacturing cost for gasoline and a 12 percent energy contribution for jet and diesel fuels. On the basis of an earlier study (Bachman et al. 1976), it appears that the consumption of energy required to produce the total pool of desulfurized gasoline will range between the equivalent of 42,000 and 175,000 barrels of crude oil per day.

Because the demand for diesel fuel may increase more rapidly than assumed in Table 4-8, two additional demand scenarios are considered (see Table 4-9). Scenario B extends Table 4-8 to include a 15 percent diesel fuel inroad into the gasoline

Table 4-8. BASE CASE SUPPLY DEMAND FORECAST
(million barrels daily)

	1976	1980	1985	1990
Mobile source demand				
Gasoline	7.02	7.48	7.24	7.00
Mobile source diesel fuel	0.81	0.97	1.28	1.76
Jet fuel	0.99	1.11	1.27	1.48
Total	8.82	9.56	9.79	10.24
Total supply forecast				
Domestic crude oil	8.1	9.0	9.7	9.8
Natural gas liquids	1.6	1.4	1.4	1.4
Foreign crude oil	5.3	6.7	7.0	7.5
Product imports	2.0	2.6	2.6	2.6
Total	17.0	19.7	20.7	21.5

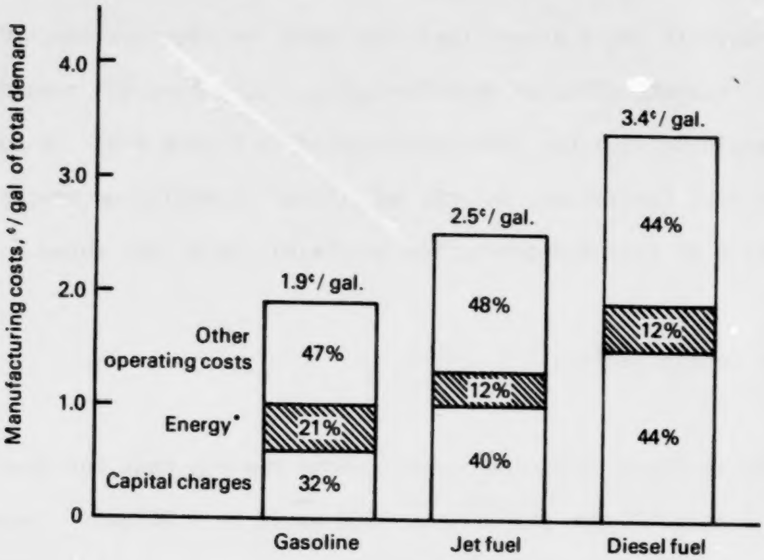
Sources: Petroleum Industry Research Foundation, Inc. (1977).
Department of Energy data on segregation of diesel fuels between mobile and stationary sources.

Table 4-9. ECONOMIC IMPACT OF SULFUR REDUCTION OF MOBILE SOURCE FUELS: 1977 COST BASIS, BASE CASE DEMAND SCENARIO

	Capital investment, \$ billion[a]		Annual manufacturing cost, \$ billion[b]	
Fuel	1985	1990	1985	1990
Gasoline	4.0	4.0	2.1	2.1
Jet fuel	0.8	1.0	0.5	0.6
Diesel fuel	1.2	1.6	0.7	0.9
Total	6.0	6.6	3.3	3.6

[a] Capital investment includes direct process investment for desulfurization, hydrogen manufacture, sulfur recovery facilities, associated off-site costs, interest during construction, and working capital. With cost escalation, final costs will be twice those estimated above.

[b] Manufacturing cost includes return on direct capital investment at a 12 to 15 percent after-tax basis, fuel (energy) costs, and labor costs. Cost escalation exceeding general inflationary rate is likely; therefore, final manufacturing costs also will exceed those estimated above. Costs for an individual refinery may vary markedly with the processing configuration and size of refinery, sulfur content of crude oil processed, and availability of surplus hydrogen.



*Estimated energy consumption (on fuel-oil-equivalent basis) directly related to sulfur reduction: gasoline, 175,000 barrels/day; jet fuel, 30,000 barrels/day; diesel fuel, 50,000 barrels/day. Includes fuel burned and electricity and steam consumed, with byproduct credits. For gasoline, desulfurization will improve efficiency of conversion of the FCC feedstock, and additional credits should be included.

Figure 4-4. Unit manufacturing costs in 1990 for sulfur reduction in mobile source fuels.

market, or a daily demand in 1990 of 5.95 million barrels of gasoline and 2.81 million barrels of diesel fuel. Scenario C forecasts a 30 percent penetration by 1990, corresponding to a daily demand of 4.9 million barrels of gasoline and 3.86 million barrels of diesel fuel. Because desulfurizing diesel fuel entails higher manufacturing costs (more diesel fuel than gasoline requires desulfurization; see Figure 4-4), separate gasoline desulfurization facilities and expanded diesel fuel refineries would inflate the investments noted in Figure 4-5. In addition, only the costs of desulfurization, not the additional marketing or processing costs associated with an expanded production of diesel fuels, are noted.

4.4.4 Other Considerations

As shown in Figure 4-6, the industry-wide average cost for desulfurizing gasoline to meet a 100-ppm standard by 1990 would be 1.9¢/gal. These costs include the costs of hydrotreating the feedstock of refinery catalytic cracking units. However, within the industry, desulfurization costs would vary widely among refiners, depending upon differences in the sulfur content of the crude oil processed, refinery capacity, and the availability of hydrogen (used in the desulfurization process).

Table 4-10 depicts typical manufacturing costs for refining three different crude feedstocks at two refineries differing in production levels--a 100,000 barrel-per-day FCC unit and a 10,000 barrel-per-day unit. Two variants of hydrogen availability are also considered: one case considers the cost of hydrogen available at fuel prices; another includes costs for construction of plant for hydrogen production.

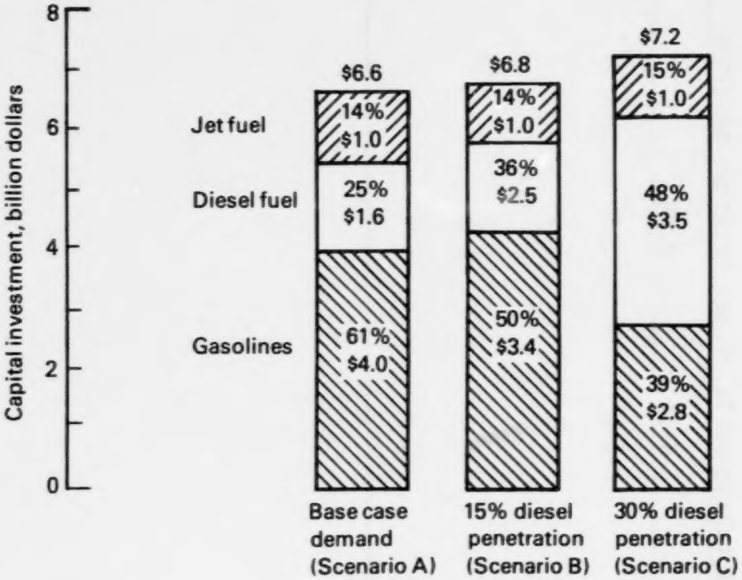


Figure 4-5. Capital investment requirements by 1990 for sulfur reduction in mobile source fuels,* 1977 cost basis.

*Not included: investments for new processing facilities to expand diesel fuel volumes to levels of Scenarios B and C; investment in existing gasoline production facilities which are not needed to supply lower gasoline demand levels of Scenarios B and C.

Table 4-10. ILLUSTRATIVE REFINERY MANUFACTURING COSTS
FOR SULFUR REDUCTION OF GASOLINE POOL,
1977 COST BASIS

	Unit manufacturing costs, ¢/gal of total gasoline		
Sulfur content of FCC feed, wt%	0.2%	0.5%	2.8%
Large refinery with fuel hydrogen	0	1.0	1.6
Large refinery with synthetic hydrogen	0	1.3	2.1
Small refinery with fuel hydrogen	0	1.6	2.5
Small refinery with synthetic hydrogen	0	2.3	3.6

A refinery processing mainly low-sulfur (sweet) crude to meet a 100-ppm sulfur level would entail no additional processing requirements. In the worst case considered in Table 4-10, a small refinery processing high-sulfur (sour) crude and building a facility for hydrogen production could incur twice the costs shown.

Higher costs would probably be borne with greater difficulty by small refineries than large ones. Thus, the economic advantage and competitive status of larger refineries could increase. Among other considerations, differences in the incremental costs for desulfurization may allow refiners with comparatively low desulfurization costs to compete in geographic markets now unavailable to them. That is, the cost of transporting materials to more distant markets would be overcome by their relatively lower total production costs. The impact of this situation could be profound (see Figure 4-6) in the Rocky Mountain (PADD IV) region, where small refineries process crudes that would incur relatively high costs for desulfurization. Analogous trends exist also for diesel and jet fuels. Also, it should be noted that refineries do not have complete freedom to select low-sulfur crudes for any particular refining-processing scheme. Most refineries cannot segregate products of one crude from products of another crude. Another problem not specifically addressed is that some low-sulfur crudes are highly paraffinic, creating a pour-point problem in diesel fuels, and would require additional desulfurization.

Finally, new refineries take several years to build--1 or 2 years for design, permits, and preliminary environmental monitoring and 2 to 3 years for construction and start-up, assuming no environmental prohibitions on new construction. Consequently, sulfur regulations on all three fuels could probably not be implemented until 1985. In light of these difficulties, refiners might import additional sweet crudes, even though they are more expensive.

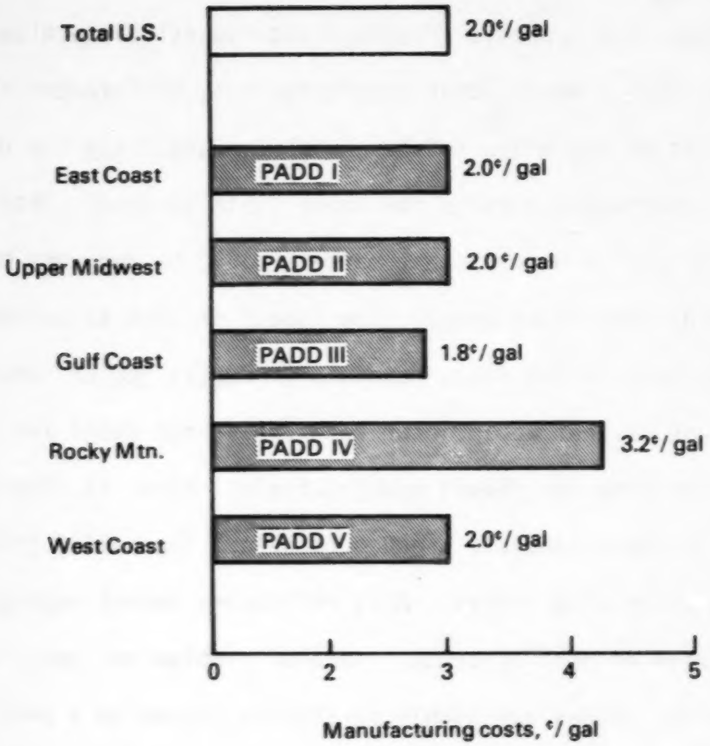


Figure 4-6. Regional manufacturing costs* of sulfur reduction in gasoline, 1977 cost basis.

*PADD IV is comprised primarily of small refiners incurring relatively high costs. The lower costs in PADD III reflect the availability of sweet crude oil. It should be noted that Alaskan North Shore oil is relatively high in sulfur.

4.4.5 Further Action Needed

To assess more fully the impact of manufacturing low-sulfur, mobile source fuels, additional considerations not possible in this study should be given:

1. The relationship between feedstock sulfur content and gasoline sulfur content for FCC units and coking units should be refined to allow more accurate projections of gasoline sulfur levels and desulfurization requirements.

2. Impact estimates of producing low-sulfur jet fuel and diesel fuel should be extended to include effects of economies of scale and availability of refinery hydrogen.

3. Selected computer model simulations should be initiated which include the parameters investigated in this study, but which further allow additional processing and blending interactions to be explored in order to meet target sulfur specifications at minimum costs.

4. Certain novel processing techniques for sulfur reduction in distillate products, which are not yet commercially proven, should be evaluated to determine cost reductions possible through likely technological innovations during the next decade.

5. The impact of sulfur control of mobile source fuels should be assessed more fully for small refiners processing less than 50,000 barrels of

crude oil per day. Such studies should include examinations of the likely effect of regulations on the competitive structure of the industry.

6. Since imposition of sulfur regulations on the mobile source fuels could increase the cost of low-sulfur crude oils to U.S. refiners, the supply and demand for low-sulfur crude oils should be examined, including opportunities for rationalization of crude oil distribution for optimal fuel production.

4.5 SHORT-TERM ALLOCATION OF LOW-SULFUR CRUDE OIL

As noted in the preceding section of this chapter, desulfurization of fuel is one possible means to reduce sulfate emissions. Implementation of this or some other technological control alternative, however, could take years. In the meantime, while alternate controls are brought about, sulfate emissions could be controlled by refining fuel from crude oils with an inherently low sulfur content. Thus, this section, written by J. M. Kawecki and S. L. Smith, considers the option of short-term allocation of low-sulfur crudes--those containing less than 0.5 percent sulfur.

For purposes of discussion, Bureau of Mines (BOM) District 13 will be considered in a scenario where such an allocation might be made. California, the major oil refiner in District 13, currently has the highest vehicular sulfate emissions of any State. Not only do the California automotive oxidation catalysts required for HC and CO control emit higher sulfate levels, but the gasolines themselves, particularly those refined in southern California, historically contained higher amounts of sulfur on the average than gasolines refined elsewhere in the United States.

Consequently, the implementation of sulfate control alternatives would be especially relevant to this area.

To understand the impact of allocating low-sulfur crudes to California, several factors should be considered. First, California already regulates unleaded-grade gasoline sulfur levels to 400 ppm. In addition, a 300-ppm limit goes into effect in 1980. Recently, the California Air Resources Board was petitioned to rescind this 1980 regulation.

Second, California supplemented its domestic crude stocks with foreign imports. From 1970 to the summer of 1977, foreign imports rose from 23 to 54 percent of the total crudes processed in BOM District 13. A slight decline in foreign imports occurred in the second half of 1977, reflecting the entry of Alaskan North Slope oil. Roughly half of these imports were from Indonesia, a source of relatively low-sulfur crude oil. Projections indicate that by 1985, increasing supplies of Alaskan oil into District 13 may reduce foreign oil imports. Most of the Alaskan crude is sour, having an average sulfur content of 0.9 percent. Thus, over the next decade, there may be a marked increase in the processing of high-sulfur crudes.

Because of current regulations on the sulfur content of fuel oil, almost all low-sulfur crudes are processed for fuel oil (with a 25-50 percent yield). Since most existing facilities cannot desulfurize sour crude oils to meet the 0.25 percent sulfur limit for residual oil, there will be additional demands for low-sulfur crudes. While Federal pricing regulations allow refiners to pass on all operating costs, including depreciation, to consumers, they cannot receive additional returns for their initial capital investments in constructing desulfurization facilities.

Consequently, it appears that almost all of the low-sulfur crudes will continue to be used for residual oil in the future. The exact impact of this anticipated trend is difficult to predict. However, the trend of lower sulfur levels found in southern California's unleaded-grade gasoline, currently averaging 290 ppm, will probably reverse unless new desulfurization facilities are added or unless foreign imports of low-sulfur crudes are increased. Nevertheless, if unleaded gasoline sulfur levels remain about the same through 1985, vehicular sulfate emissions for southern California will probably not change significantly, assuming that emission control devices that emit large amounts of sulfuric acid (e.g., TWC plus air and oxidation catalysts) are not used in the 1980 model vehicles.

According to the National Energy Plan, U.S. gasoline demand for transportation use is expected to decrease from a 1980 peak by about 5.2 percent by 1985. This decrease will be offset by an increase in diesel fuel, which contains two to three times as much sulfur as gasoline. However, the smaller amount of sulfate emitted by diesel engines might tend to balance out the sulfate emitted by the percent increase of oxidation catalyst-equipped vehicles.

Finally, if this short-term allocation scenario is applied to the entire Nation, there may not be enough low-sulfur crude oil at acceptable prices to meet the transportation demand. Naturally, California would be hardest hit in this scenario, in view of its current sulfur regulations.

Generally, the short-term allocation of low-sulfur crude oils does not appear to represent a viable control alternative. At present, these oils are available from only three main sources--one domestic (midcontinent, except west Texas) and two foreign (Malaysia and Africa). Not only are these oils expensive, but their supply is so competitively limited that refiners would still have to rely heavily on the more plentiful sour crudes (e.g., Alaskan North Slope crude) to meet fuel production demands. Hence, a short-term allocation of low-sulfur crude oils would be costly and have little impact on mobile source emissions for the State of California. Moreover, California is already increasing its imports of low-sulfur crude, while roughly 35 percent of the Alaskan North Slope oil is being shipped through the Panama Canal to other refining districts. The increased use of imported oils (from Indonesia and Nigeria) would run counter to the National Energy Plan, which stresses reliance on domestic fuel reserves.

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5. COST/BENEFIT ANALYSIS

This chapter summarizes parts of a study by Lee W. Merkhofer et al. of SRI International and J. W. Kawecki of Biospherics, Inc., that was designed to analyze the relative costs and benefits of controlling sulfur-bearing compounds emitted from mobile sources. Although the emphasis is on sulfates, sulfur dioxide is also discussed. This analysis presents an assessment of exposure to sulfate compounds emitted from mobile sources; however, no health effects from these exposures can be determined at this time.

The assessment is presented for two scenarios. The first involves in-roadway modeling studies to determine exposures to vehicle drivers and persons close to the line source. The models, which are derived directly from emissions data, include a worst case for sulfate and sulfur dioxide exposures. The second scenario involves an areawide, incremental contribution from mobile sources in the Los Angeles Basin and the Kansas City, Mo., metropolitan area. A third scenario was attempted for a city street canyon, as found in most large cities, but there were no validated models available for this assessment.

In addition to these two assessments, the effects on the public welfare are quantified in the analysis of the costs and benefits of control for the Los Angeles Basin. Although the cost/benefit analysis for welfare effects and the exposure assessment for health are based on limited information, even a rough projection of the extent to which new catalyst technologies and other control devices might mitigate the consequences of sulfur pollution should be helpful to policymakers both in formulating standards and in setting priorities for further research.

5.1 IN-ROADWAY MODELING STUDIES

Three roadway modeling-measuring studies are discussed: the General Motors Sulfate Dispersion Experiment, the Los Angeles Catalyst Study, and the Los Angeles Roadway Measurement Study. A stochastic model is also included to determine worst-case exposure levels and probabilities.

5.1.1 General Motors Sulfate Dispersion Experiment

The GM study was designed to determine the relationship between automotive sulfate emissions and ambient air concentrations. During October 1975, a fleet of 350 low-mileage 1975 and 1976 vehicles equipped with catalysts and air pumps was operated in packs on four lanes on a 10-km north-south straightaway at a speed of 50 mph for 2 hr/day. The experiment simulated a highway flow rate of 5460 vehicles/hr. Eight GM vehicles were equipped to emit sulfur hexafluoride as a tracer, and one EPA vehicle was equipped to measure sulfate and sulfuric acid emission rates and particle sizes. The fuel contained 0.03 percent sulfur by weight.

The results of the test were as follows: the average sulfate emission rate was calculated as 37 mg/mile; aerosol particle diameters were generally between 0.01 and 0.1 μm; and more than two-thirds of the sulfate emitted by the vehicles was measured as sulfuric acid at a distance of 20 m from the test track.

Twenty meters downwind of the roadway, average sulfuric acid concentrations ranged from 1.1 to 5.4 μg/m^3. Measurements made from towers equipped with sampling devices at heights between 0.5 and 9.5 m showed that sulfate concentrations were highest near the roadway and decreased rapidly with distance. Peak sulfate concentrations, averaging 8 μg/m^3, were measured at a height of 0.5 m at median and roadside towers. Samples taken farthest (100 m) from the roadway indicated a partial neutralization of the sulfuric acid. Sulfate concentrations averaged 4 μg/m^3 inside the test vehicles and 5.2 μg/m^3 in the roadway median.

Comparison between computations using the HIWAY model (which simulates vehicle emissions on a highway by summing several point sources to integrate numerically the Gaussian point source equation over a specific distance) and these results showed agreement when meteorological conditions were unstable and the wind was nearly perpendicular to the roadway at a speed of 1 m/sec or greater. Under stable conditions or when the wind was parallel to the track, the model overpredicted the test results by a factor of 2 to 3. These results indicated that previous worst-case mobile source sulfate models overpredicted concentrations by about the same factors.

5.1.2 Los Angeles Catalyst Study

The Los Angeles Catalyst Study (LACS) was designed to investigate ambient levels of sulfuric acid, sulfate aerosols, and other catalyst emission products in areas adjacent to a heavily traveled freeway.

Initial LACS summary reports indicated that freeway contributions of sulfates averaged a high of 5 $\mu g/m^3$, but it was later determined that these amounts were affected by artifact sulfate formation. Preliminary modeling results from the 1977 data indicate an average freeway contribution of about 4 $\mu g/m^3$. The data used for this analysis are not consistent with regard to sulfate collection methods and previous modeling results for 1975 and 1976.

5.1.3 Los Angeles Roadway Measurement Study

The Los Angeles Roadway Measurement Study measured sulfuric acid, other ultrafine particles, and total sulfur on the roadway. Comparison of the results of this study with off-roadway, background concentrations showed that ultrafine sulfate emissions decreased rapidly with increased odometer mileage: at 3500 odometer miles, catalyst-equipped vehicles emitted 7 mg/mile H_2SO_4 during the FTP driving cycle and 22 mg/mile during the CUE driving cycle; at 12,550 miles, the corresponding emissions were 2.6 and 10.5 mg/mile. Worst-case emission estimates showed that 1 percent of catalyst-equipped vehicles with fewer than 4000 miles emitted 100 mg/mile H_2SO_4, while 5 percent of these vehicles emit more than 50 mg/mile H_2SO_4. A recent report by Dzubay et al. (1978) indicates that the in-roadway concentrations of sulfate particles collected during this study averaged less than 1.5 $\mu g/m^3$.

5.1.4 Worst-Case Model

Typical exposure estimates do not usually include extreme conditions, which are useful in developing policies to protect the public health and welfare. Consequently, this section presents a worst-case scenario for mobile source emissions of sulfuric acid and sulfur dioxide, using data derived primarily from a study by Papetti and Horowitz (1975); their report was amenable to linear scaling to allow for different emission rates. A scenario of this type is based on the simultaneous occurrence of rare events, such as prolonged periods of travel on high-volume freeways under meteorological conditions conducive to high pollutant concentrations. Furthermore, sulfuric acid exposure probabilities are based on the assumption that all the vehicles are equipped with catalysts, while sulfur dioxide exposure probabilities assume that all vehicles are equipped with diesel engines.

In this section, the short-term exposures (1-hr average) are estimated for a particular urban subpopulation: weekday morning, peak-period travelers in Los Angeles. According to Tiao et al. (1974), meteorological and traffic conditions between 6 and 9 a.m. tend to cause above-average concentrations of sulfur compounds. The travelers during this period include roughly 30 percent of the total Los Angeles population.

Two types of data, travel and meteorological, were used in constructing these exposures. Travel data were obtained from the Los Angeles Regional Transportation Study (LARTS), which included 36,733 weekday morning, peak-period trips along with estimated time spent on city streets and freeways during each trip. Meteorological data from Los Angeles International Airport included wind speed, wind angle, and stability class during the morning peak period for 1964. The authors noted that meteorological conditions for that year were not conducive to either high or low pollutant concentrations.

Mean travel times are short for most travelers, but are long for a few. For example, 55 percent of the peak-period travelers do not use freeways during the hour following the start of the morning, peak-period trip; only 1 percent of the travelers use freeways for at least 56 min during the hour following the start of the trip (see Table 5-1).

The joint frequency distributions of the 1-hr average concentrations of sulfuric acid and sulfur dioxide were derived by modifying the HIWAY diffusion model. Traffic volumes of 3200 vehicles/hr on city streets and 17,000 vehicles/hr on freeways were used, corresponding to peak hour traffic flows in Los Angeles in 1975. In was assumed (1) that sulfuric acid and sulfur dioxide were not removed from the atmosphere during the modeling period and (2) that catalyst equipped vehicles emitted 5 and 17.5 mg/m^3 H_2SO_4 on city streets and freeways, respectively, and that diesel-powered vehicles emitted 450 mg/mile SO_2.

As seen in Table 5-1, the peak 1-hr average concentrations on freeways could be as high as 145 $\mu g/m^3$ for H_2SO_4 and 1800 $\mu g/m^3$ for SO_2. Ninety-nine percent of the time, concentrations would be less than 20 $\mu g/m^3$ H_2SO_4 and less than 500 $\mu g/m^3$ SO_2. On city streets, less than 2 $\mu g/m^3$ H_2SO_4 could occur 99 percent of the time.

Table 5-1. FEATURES OF THE DISTRIBUTION OF TRAVEL TIME DURING THE HOUR FOLLOWING THE START OF A MORNING PEAK-PERIOD TRIP IN LOS ANGELES

	Time on streets, min	Time on freeways, min	Total travel time, min
Average	11	6	17
Median	9	0[a]	13
95th percentile	29	32	52
99th percentile	43	56	60
Maximum	60	60	60
Probability of maximum	0.001	0.008	0.03

[a] 55 percent of peak-period travelers do not use freeways during the hour following the start of the trip.

For fixed values of city street and freeway travel times and of sulfuric acid and sulfur dioxide concentrations, exposure during the hour following the start of a peak-period trip was computed (Table 5-2).

Table 5-2. FEATURES OF THE DISTRIBUTION OF CONCENTRATIONS OF AUTOMOBILE-EMITTED SULFURIC ACID ON STREETS AND FREEWAYS IN LOS ANGELES

	Concentration, µg/m³[a]			
	City streets		Freeways	
	H_2SO_4	SO_2	H_2SO_4	SO_4
Average	0.5	45	3.5	90
Median	<1[b]	<90	<2.5	<60
95th percentile	<1[b]	<90	<9.5	<240
99th percentile	2	<180	<20	<500
Maximum	11	990	145	∿1800
Probability of maximum	8 x 10-5		8 x 10-5	

[a] Concentrations represent increments over background, 1-hr average.
[b] 1 µg/m³ H_2SO_4 is the 98th percentile.

As seen in Table 5-3 and Figures 5-1 and 5-2, the 99th percentile of exposure is 6 μg/m^3 H_2SO_4 and 150 μg/m^3 SO_2, while the median value is 1 μg/m^3 and H_2SO_4 and less than 35 mg/m^3 SO_2. These data are based on approximately 109 morning peak-period trips in Los Angeles (1967 data).

Using the variables in this exposure scheme, the following conclusions can be drawn:

1. The median 1-hr exposure will be <1 μg/m^3 H_2SO_4 and <25 μg/m^3 SO_2.
2. The average 1-hr exposure will be 1 μg/m^3 H_2SO_4 and 25 μg/m^3 SO_2.
3. The 99th percentile of 1-hr exposures will be 6 μg/m^3 H_2SO_4 and 150 μg/m^3 SO_2.
4. The maximum 1-hr exposure will be 145 μg/m^3 H_2SO_4 and 2600 μg/m^3 SO^2 and will occur with a probability of 6×10^{-7}.

The probability of reaching the maximum exposure is 1 hr out of approximately every 200 years.

5.2 AREAWIDE STUDIES

These studies show that roadway emissions disperse rapidly and are not likely to cause high localized levels on or near the roadway. The

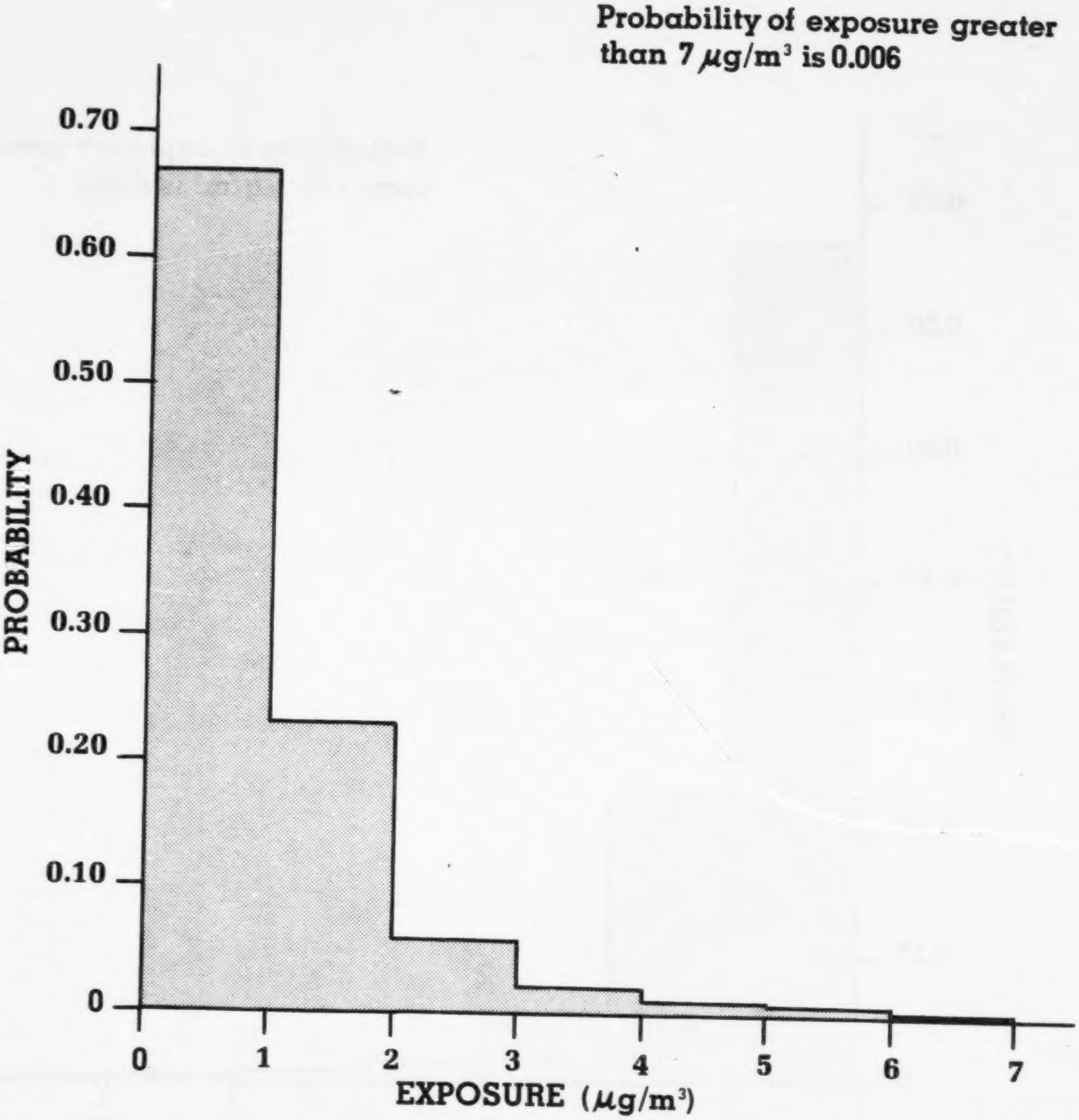


Figure 5-1. Histogram of sulfuric acid exposure.

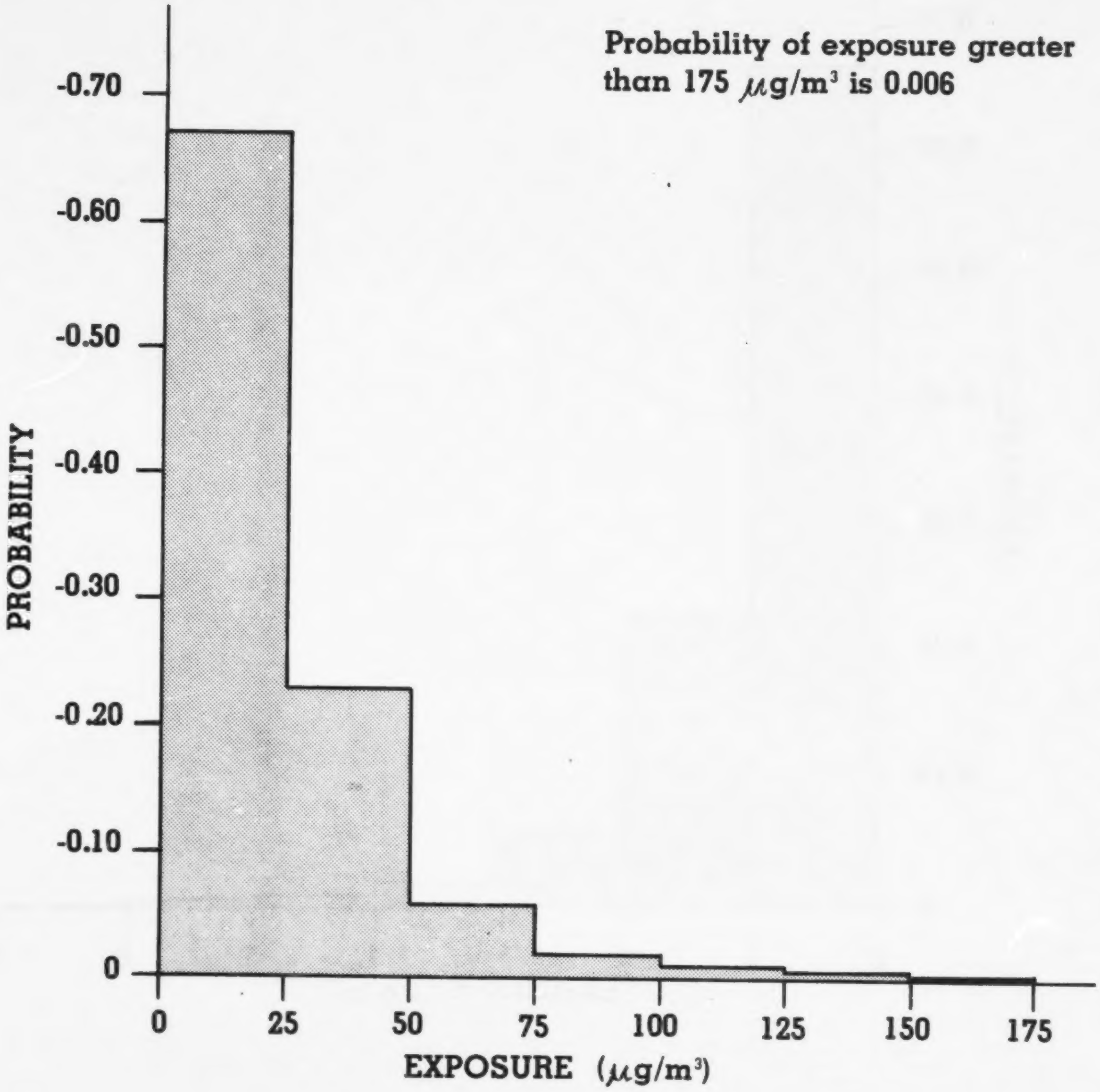


Figure 5-2. Histogram of sulfur dioxide expsoure.

Table 5-3. FEATURES OF THE DISTRIBUTION OF 1-HR AVERAGE EXPOSURES TO AUTOMOBILE-EMITTED SULFURIC ACID DURING THE HOUR FOLLOWING THE START OF A PEAK-PERIOD TRIP IN LOS ANGELES

	Exposure, μg/m³	
	H_2SO_4	SO_2
Average	1	25
Median	<1[a]	<25
95th percentile	3	75
99th percentile	6	150
Maximum	145	2600
Probability of maximum	6 x 10-7	

[a] 1 μg/m³ is the 67th percentile.

incremental contribution on background ambient air levels could, however, be significant. While mobile sources account for only about 2 percent of the total anthropogenic sulfur emissions nationwide, in some areas of the country this percentage would be higher. The Los Angeles Basin and the Kansas City, Mo., metropolitan area will be used in this areawide scenario. A limitation inherent in assessing an areawide contribution from mobile source sulfur-bearing compounds is that the process by which sulfur emissions contribute to atmospheric sulfur levels is complex and not fully understood. Sulfur dioxide (SO_2), the primary sulfur compound emitted by fossil fuel combustion, reacts chemically in the ambient air to form sulfates. Some of these sulfate particles are known to be more toxic than SO_2 and have been associated with adverse health effects, visibility reduction, and nonbiological and biological materials damage.

5.2.1 Los Angeles Basin Study

The Los Angeles Basin is a geographic area where high mobile source sulfate and sulfur oxide emissions could have the greatest areawide effect. Sulfate levels measured in the Los Angeles Basin are unusually high considering the area's total sulfur oxide emissions. This anomaly is attributed to the basin's meteorological and geographic characteristics, which are extremely favorable to the buildup of $SO_4^{=}$. Frequent temperature inversions, low average wind speeds, and the surrounding mountains limit air movement. The abundant sunshine and photochemical smog are believed to promote an unusually rapid and complete conversion of SO_2 to $SO_4^{=}$. Furthermore, limited rainfall precludes frequent clearing of sulfates from the atmosphere.

Two factors contribute to concern that future sulfate levels in Los Angeles will be even higher than they are today. First, forecasts of sulfur oxide emissions to 1980 indicate significant increases because of shortages of relatively clean natural gas and increased burning of sulfur-containing fuel oil (Hunter and Helgeson 1976). Thus, sulfur compound levels can be expected to increase because of increased SO_2 emissions from stationary sources.

The second factor causing concern is related to automobile emissions. Beginning with the 1975 model year, automobile manufacturers began installing oxidation catalysts on cars as a means of reducing emissions of carbon monoxide (CO) and hydrocarbons (HC). Without a catalytic converter, sulfur contained in gasoline passes out of the tailpipe mainly as SO_2. By contrast, cars equipped with oxidation catalysts tend to convert part of the SO_2 directly into sulfur trioxide, which rapidly combines with water vapor in the exhaust to form sulfuric acid aerosol. Sulfuric acid is known to be one of the more harmful forms of sulfate. As more stringent standards for CO and HC take effect and as older cars are removed from the vehicle fleet, there is concern that the high density of Los Angeles automobile traffic will result in large local sulfate concentrations.

5.2.1.1 Methods for Reducing Automobile Sulfur Emissions-- Several approaches that reduce sulfuric acid and still meet the more stringent standards established for other pollutants are being investigated. The obvious way to avoid the sulfate problem is to desulfurize gasoline. Sulfur dioxide and sulfate emission levels are directly proportional to fuel sulfur level. Therefore, reducing the level of sulfur in the fuel will reduce sulfate as well as SO_2 emissions. This approach would require the oil industry to invest substantial capital in equipment and reduce the net amount of gasoline energy obtained per barrel of crude oil. However, there is general agreement that most of the sulfur in today's gasoline could be removed by further refining.

Another approach is to use chemical sulfate traps to remove particulate matter from the exhaust. Some of the designs tested have proven to be quite effective. However, a number of problems must be solved before a practical design exists for vehicle application. The traps tend to be fragile and are easily damaged by engine failures. Problems have also occurred with the trap causing excessive exhaust pressure buildup, which causes a loss in automobile fuel economy.

The approach gaining the most attention is to redesign the catalyst in such a way as to limit the production of sulfates. Experiments with catalysts show that their chemical formulation, physical shape, temperature, oxygen pressure, and age all affect the production of sulfuric acid. The most important of these appears to be the oxygen level in the exhaust gas to the catalyst: pumping air into the catalyst (to improve the efficiency of HC and CO removal) generally results in more sulfuric acid. One of the most promising alternative designs is the three-way catalyst. With this approach, the fuel/air mixture to the engine is maintained precisely at the stoichiometric point (enough air to burn all HC and CO, but not more than enough). To maintain the proper mixture ratio, a feedback control is normally employed with an oxygen sensor in the exhaust. If an oxidation catalyst is added to the three-way catalyst, the system is capable of meeting even the low 1980 standards for HC and CO with low $SO_4^=$ emission. However, the complexity of the approach is a drawback. Controlling the air injection rate to the oxidation catalyst will probably require additional sensing and control devices (Office of Mobile Source Air Pollution Control 1977).

For each control category, four levels of control were considered: (1) a minimum control level, little or no effort to reduce emissions; (2) a base case; (3) a moderate level; and (4) a maximum control level, theoretical, maximum reduction possible. Reducing the sulfur content of the fuel was assumed to apply uniformly to the entire vehicle fleet. Reductions in SO_2 to $SO_4^=$ conversion in the exhaust and the trapping of $SO_4^=$ emissions were assumed to affect the 1979 and 1980 vehicles only.

5.2.1.2 Emissions Policies Investigated Analysis--The control methods mentioned above (as well as the several other approaches that are being investigated) offer varying degrees of promise for reducing $SO_4^=$ emissions while allowing the levels of other emissions to be reduced still further. All proposed methods will impose added costs on the drivers of automobiles through decreased auto performance, higher fuel consumption, or higher operating and purchase costs.

Techniques for sulfur emissions control are still in an early stage of development, and it is difficult to predict either the ultimate costs of the controls or their efficiency under real-world driving conditions. Rather than attempt to estimate the ultimate costs and effectiveness of the various devices that are currently being researched and developed, this analysis investigated the benefits of achieving various reductions in average SO_2 and $SO_4^=$ emissions from the automotive fleet mix for the year 1980.

Table 5-4 summarizes the specific set of assumptions for which benefits were calculated. The first control category considered was reduction of the sulfur content of the fuel. As stated above, reducing the sulfur in the fuel proportionally reduces both SO_2 and $SO_4^=$ emissions. The second type of control considered was reduction in the percent conversion in the exhaust of SO_2 to $SO_4^=$. Improved catalysts have the effect of reducing $SO_4^=$ emissions while proportionally increasing SO_2 emissions. The third control considered reducing $SO_4^=$ while keeping SO_2 constant. Such an effect is characteristic of "traps."

Table 5-4. AUTOMOBILE EMISSION ASSUMPTIONS EVALUATED BY ANALYSIS

Control approach	Minimum control assumptions			Base case assumptions		
	Percent fuel sulfur	Percent conversion[a]	Emissions,[a] mg/mile	Percent fuel sulfur	Percent conversion[a]	Emissions,[a] mg/mile
Reduce fuel sulfur	0.07	b	b	0.05	13.8	25.0
Reduce[c] conversion in exhaust	b	27.6	b	0.05	13.8	25.0
Trap[c] $SO_4^=$ emissions	b	b	25.0	0.05	13.8	25.0

Control approach	Moderate control assumptions			Maximum control assumptions		
	Percent fuel sulfur	Percent conversion[a]	Emissions,[a] mg/mile	Percent fuel sulfur	Percent conversion[a]	Emissions,[a] mg/mile
Reduce fuel sulfur	0.01	b	b	0.00	b	b
Reduce[c] conversion in exhaust	b	6.9	b	b	0.0	b
Trap[c] $SO_4^=$ emissions	b	b	12.5	b	b	0.0

[a] Percent conversion and $SO_4^=$ emissions are stated as they would be measured under standardized conditions of freeway driving: 0.03 percent sulfur fuel, and 13.6 miles/gal vehicle gas mileage. The values stated are for 1980 vehicles. For 1979, emissions are 80 percent of the values shown.

[b] Same entry as base case assumption.

[c] Control affects 1979 and 1980 model years only.

For the cost/benefit analysis, the model region chosen was a square about 85 km on each side overlying the central portion of the Los Angeles Basin as shown in Figure 5-3. The region was subdivided by a grid into 169 cells, each roughly 4 miles on a side. (This square region is approximately 2 miles/side larger than the same region modeled by Cass [1978] and Roth [1974]. The overlap allows us to make use of data and results of these authors.) Spatial distribution was obtained by individually estimating concentrations within each cell. Mobile emissions within a cell were the sum of four categories: automobile (including light trucks), heavy trucks, aircraft, and ships.

Automobile emissions were assumed to be proportional to local traffic density. Estimates of the spatial distribution of freeway and surface street traffic for 1969 derived by Roth (1974) were used to estimate the daily vehicle miles traveled within each cell, as shown in Figures 5-4 and 5-5.

From these assumptions, the average emission rates for the 1980 on-the-road vehicle fleet were estimated. Estimates were performed separately for surface and freeway driving, assuming a vehicle age and use distribution given in Nordsieck (1975, p. 19). All sulfur in gasoline that was not converted to $SO_4^=$ was assumed to be emitted as SO_2. The resulting average emission rates obtained are summarized in Table 5-5.

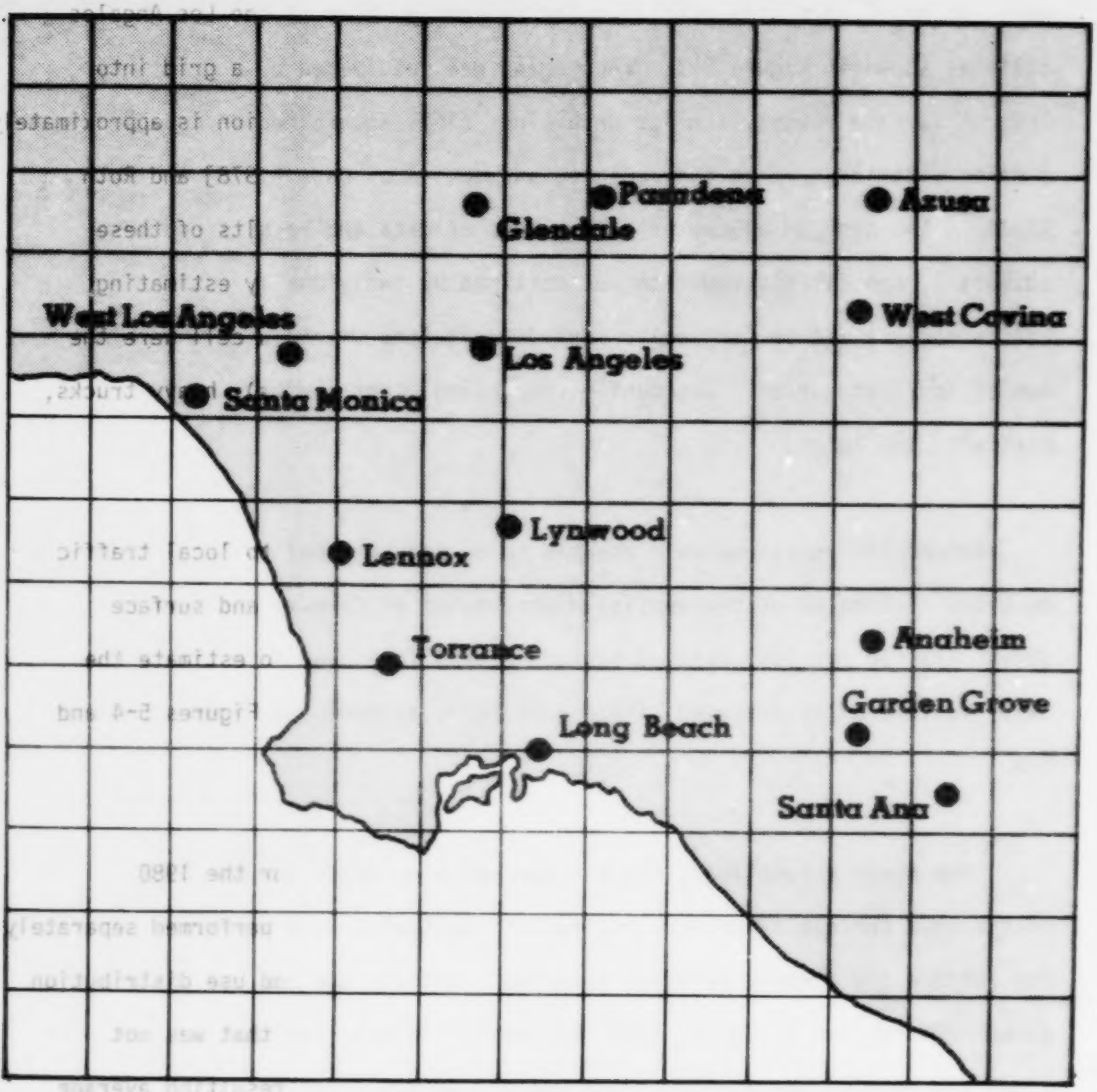
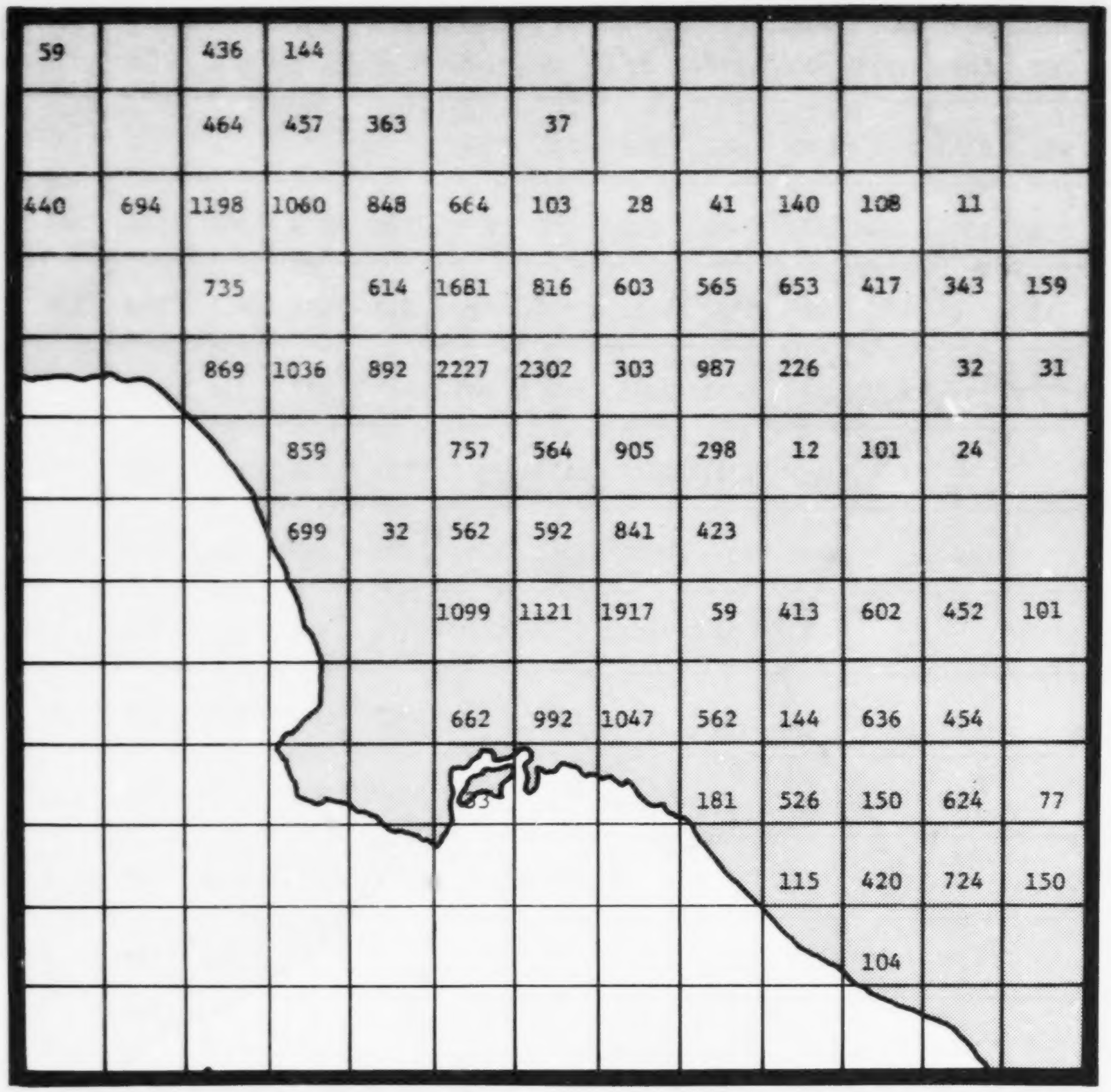

Pasadena
Azusa
Glendale
West Los Angeles
West Covina
Los Angeles
Santa Monica
Lynwood
Lennox
Anaheim
Torrance
Garden Grove
Long Beach
Santa Ana

Figure 5-3. Modeling region and cells defined for estimation of SO_4= levels.



59		436	144									
		464	457	363		37						
440	694	1198	1060	848	664	103	28	41	140	108	11	
		735		614	1681	816	603	565	653	417	343	159
		869	1036	892	2227	2302	303	987	226		32	31
			859		757	564	905	298	12	101	24	
			699	32	562	592	841	423				
					1099	1121	1917	59	413	602	452	101
					662	992	1047	562	144	636	454	
					83			181	526	150	624	77
									115	420	724	150
										104		

Figure 5-4. Spatial distribution of freeway traffic,1969.

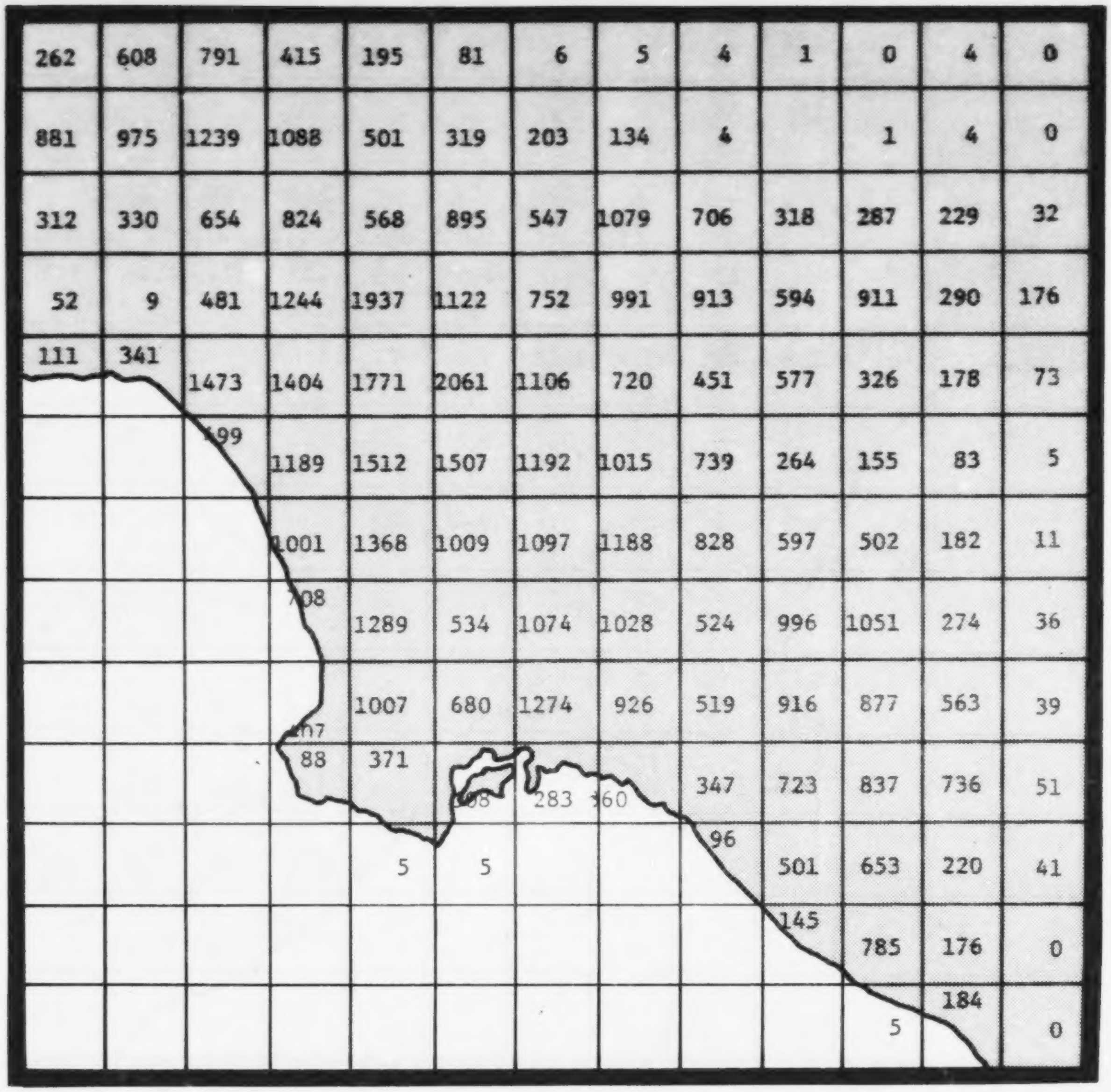

262 608 791 415 195 81 6 5 4 1 0 4 0
881 975 1239 1088 501 319 203 134 4 1 4 0
312 330 654 824 568 895 547 1079 706 318 287 229 32
52 9 481 1244 1937 1122 752 991 913 594 911 290 176
111 341 1473 1404 1771 2061 1106 720 451 577 326 178 73
499 1189 1512 1507 1192 1015 739 264 155 83 5
1001 1368 1009 1097 1188 828 597 502 182 11
708 1289 534 1074 1028 524 996 1051 274 36
407 1007 680 1274 926 519 916 877 563 39
88 371 208 283 360 347 723 837 736 51
5 5 96 501 653 220 41
145 785 176 0
5 184 0

Figure 5-5. Spatial distribution of surface street traffic, 1969.

Table 5-5. AVERAGE FLEET $SO_4^{=}/SO_2$ EMISSION RATES FOR 1980 ESTIMATED UNDER VARIOUS ASSUMPTIONS (mg/mile)

	Minimum control assumption		Base case assumption		Moderate control assumption		Maximum control assumption	
	Freeway	Surface	Freeway	Surface	Freeway	Surface	Freeway	Surface
Reduce fuel sulfur	16.4/204	7.42/273	11.7/149	5.3/195	2.34/30	1.06/39	0/0	0/0
Reduce conversion in exhaust	18.4/145	8.1/193	11.7/149	5.3/195	8.0/152	3.7/196	5.1/154	2.5/197
Trap $SO_4^{=}$ emissions	11.7/149	5.3/195	11.7/149	5.3/195	3.9/149	5.1/195	5.1/149	2.4/195

Total SO_2 and $SO_4^=$ emission levels from automobiles were obtained by multiplying emission rates by estimates of surface and freeway vehicle traffic. The vehicle traffic estimates used for 1980 were 1974 estimates inflated to 1980, assuming growth rates by cell that were identical to the growth rates observed by Cass (1978) between 1969 and 1974.

Estimated 1980 sulfate concentrations attributable to automobiles calculated by the model range between zero and about 4 $\mu g/m^3$. Figure 5-6 shows sulfate concentrations attributable to automobile emissions under the base case assumption.

The alternative emission scenarios are summarized in Table 5-4. The estimation procedures and assumptions employed were as follows. The average $SO_4^=$ emission rate from new autos during the period 1975-78 under standardized conditions of freeway driving, 13.6 miles/gal fuel economy and 0.03 percent fuel, was assumed to be 15 mg/mile. As the car aged, emission rates were assumed to decrease by 25 percent per year because of deterioration of the catalyst. The base case emission rate assumption for 1979 and 1980 autos under the same standardized conditions was 20 mg/mile and 25 mg/mile, respectively. The assumed increase was to account for the possible difficulty in meeting more stringent CO, HC, and NO_x emissions standards for 1979 and 1980. Because of $SO_4^=$ storage on the catalyst substrate, emission rates during surface street driving were assumed to be only one-third the rate during freeway driving. Non-catalyst-equipped vehicles were assumed to emit 1 mg/mile $SO_4^=$ under both surface street and freeway driving and to comprise a constant 15 percent of each new year's vehicle fleet.

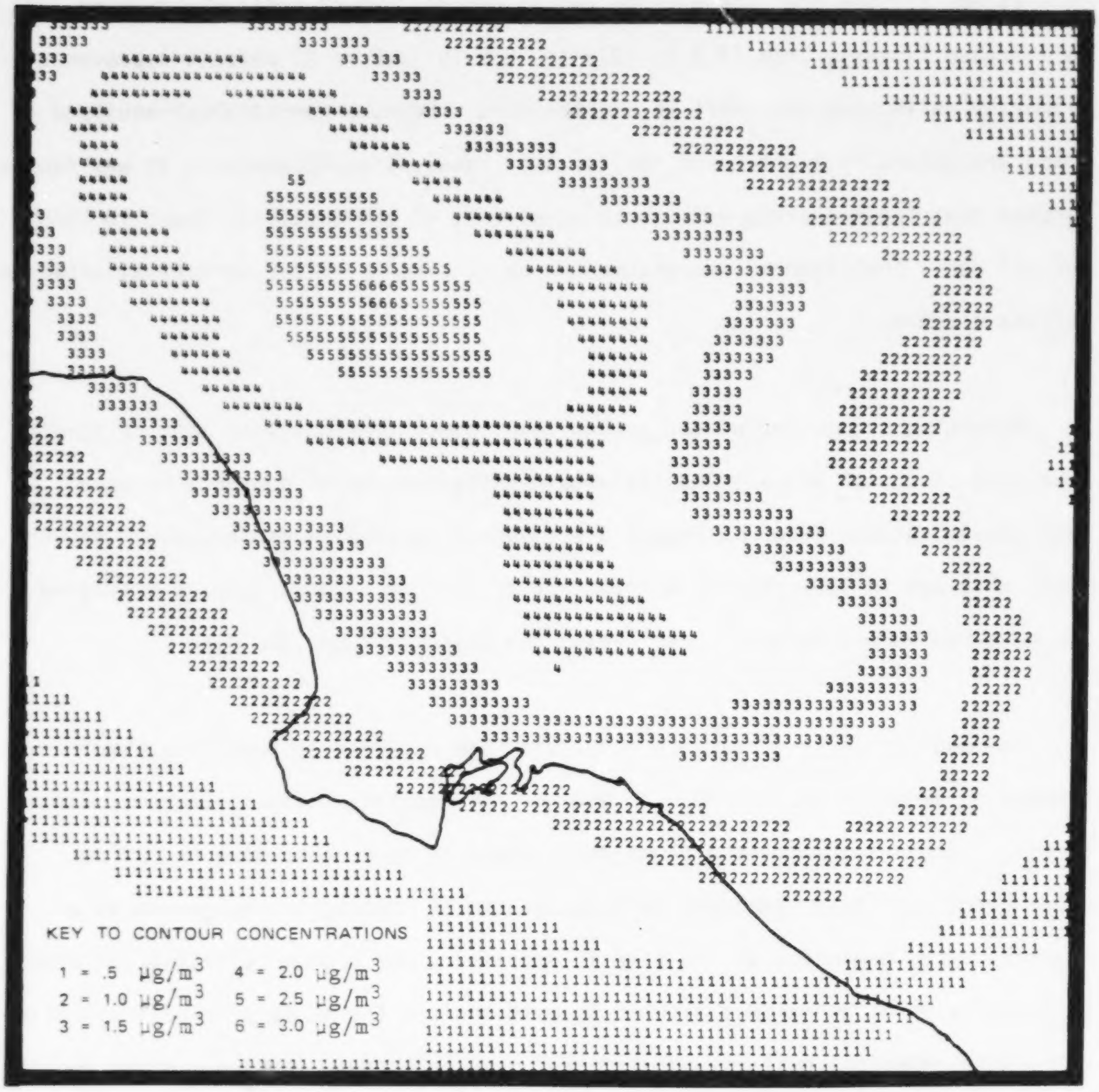
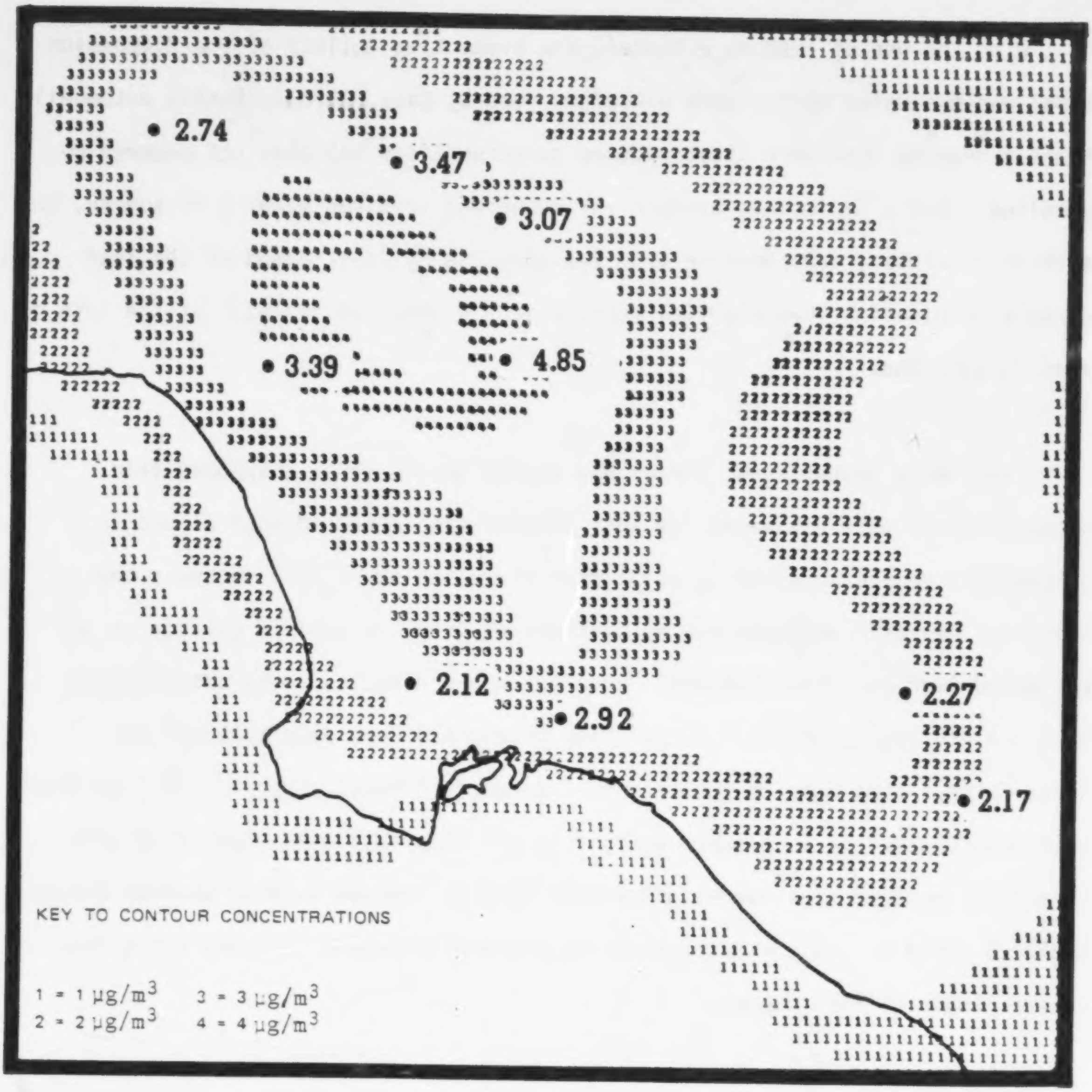

KEY TO CONTOUR CONCENTRATIONS
1 = .5 μg/m3
2 = 1.0 μg/m3
3 = 1.5 μg/m3
4 = 2.0 μg/m3
5 = 2.5 μg/m3
6 = 3.0 μg/m3

Figure 5-6. Incremental sulfate concentrations attributable to automobiles under the base case emissions assumptions, 1980.

It was assumed that the mileage (miles per gallon) of catalyst-equipped cars to increase linearly from 13.4 in 1975 to 16.5 in 1980, a 23 percent improvement primarily reflecting the shift to lighter cars. Because non-catalyst-equipped autos are generally lighter and smaller than catalyst-equipped cars, It was farther assumed that their average mileage to equal that of the catalytic fleet in 1980. For all cars, fuel economy was assumed to be 27 percent better on freeways than on surface streets.

Resulting summer and winter ground level concentrations were then integrated over wind direction and averaged to obtain estimated annual contours of equal lead concentrations shown in Figure 5-7. Annual average suspended lead measurements obtained in 1969 from nine sites are also shown. Comparison of estimated and measured values is good, considering the approximations employed.

The mobile source dispersion model was then expanded to treat the simultaneous emission of SO_2 and $SO_4^=$ to account for deposition and atmospheric conversion. Conversion within a plume was assumed to occur according to a first-order reaction. Both compounds were assumed to be removed to the ground at a constant rate, beginning at the time of emission. For a given direction of wind, each mobile point source was assumed to emit a plume the shape of a half-elliptical cone, with emissions uniformly distributed over each cross section a given distance downwind from the source. The dimensions of the plume were chosen so as to approximate downwind concentrations estimated by the binormal continuous plume dispersion model of Pasquill and Gifford (Turner 1970) for a source at ground level under atmospheric stability class C.



2.74
3.47
3.07
3.39
4.85
2.12
2.92
2.27
2.17
KEY TO CONTOUR CONCENTRATIONS
1 = 1 μg/m3
2 = 2 μg/m3
3 = 3 μg/m3
4 = 4 μg/m3

Figure 5-7. Annual average suspended lead concentrations for 1969 in the Los Angeles Basin.

With the use of lead as a tracer, the predictive ability of the dispersion model was evaluated by the same procedure used by Cass (1975). Roth's automobile traffic density data were first reduced to account for vehicles not powered by gasoline. Emission factors assumed for suspended lead (60 mg/mile in summer, 52 mg/mile in winter) were derived from the analysis by Cass, based on the lead content of gasoline, vehicle fuel economy, and a detailed analysis of the fate of lead in gasoline.

From these assumptions, summer and winter ground level suspended lead concentrations were estimated for each average wind speed in each of the 16 azimuthal directions, assuming horizontal plume angles of 22.5 degrees, and emissions per cell were derived by multiplying gasoline vehicle mileage by the estimated lead emissions factors. Wind speed and direction were obtained for each cell through a linear interpolation of monthly wind data obtained for three sites. (Los Angeles International Airport, Burbank Airport, and Long Beach. Wind data consist of wind speed and percent of time that wind blows from each direction averaged over summer and winter months. Source is U.S. Weather Bureau [1962].) Wind directions and speeds are assumed to remain constant along the entire length of each plume.

Estimated 1973 ground level $SO_4^=$ concentration for the Los Angeles Basin from all sources were obtained by adding mobile and stationary components. The 1973 mobile component was obtained by using the mobile model with 1973 automobile emission estimates, vehicle traffic estimates, vehicle traffic growth to

1973, vehicle fuel economy, and summer/winter sulfur content of gasoline. Mobile components from heavy trucks, aircraft, and ships, also taken from Cass (1978), were added to the auto component to obtain total SO_2 and $SO_4^=$ mobile emissions by cell. Using total mobile emissions, the mobile model produces contour plots for incremental $SO_4^=$ concentrations contributed by mobile sources. These mobile concentrations were then added to stationary contributions to obtain estimates of total summer/winter $SO_4^=$.

The total $SO_4^=$ estimates yielded by the combined model account for major stationary and mobile sources but neglect major sources outside of the grid, minor manmade sources on the grid (which together account for 10 percent of emissions), and natural sources. The principal natural source of atmospheric sulfur oxide is the oxidation of hydrogen sulfide or dimethyl sulfide gas that results from decaying vegetation. Near oceans, significant sulfate is also emitted as part of the sea spray. Natural background levels of atmospheric sulfate in the Los Angeles Basin have been estimated to range between 1 and 4 $\mu g/m^3$.

Background $SO_4^=$ concentrations due to sources other than the top 90 percent of stationary and mobile on-grid sources were estimated through a least-squares fit of predicted concentrations to available measurements of actual concentrations. With respective nominal estimates of 2.88 percent/hr and 1.44 percent/hr for the dry deposition of SO_2 and $SO_4^=$, respectively, average summer and winter mixing layer heights of 325 m, and a summer conversion rate of 6 percent/hr and winter conversion rate of 2 percent/hr, a best fit was obtained with respective summer and winter background concentrations of 3.3 and 2.0 μg/m^3. Figures 5-8 and 5-9 show 1973 $SO_4^=$ levels predicted by the model when background concentrations are taken into account. Totaled 1980 predicted annual emissions are given in Figure 5-10. Also shown in the figures are the summer and winter average values actually measured at various sites.

Since the model accounts for the spatial distribution of only the top 90 percent of anthropogenic sources, local impacts of small sources are not taken into account in the estimates. Consequently, local measurements may differ significantly from those predicted by the model. Nevertheless, predicted values compare reasonably well with actual measurements.

The stationary and mobile source dispersion and conversion models used to estimate the 1973 concentrations shown in Figures 5-8 and 5-9 are used in the analysis to predict 1980 $SO_4^=$ concentrations required by the cost/benefit analysis. The only variation was the use of projected 1980 emissions rather than 1973 emission estimates. In the absence of better information, emission rates were assumed to change uniformly irrespective of spatial distribution within each source category. Nonautomotive mobile emissions are projected to increase uniformly with a growth rate of 2 percent year.

5.2.1.3 Visibility in the Los Angeles Basin--It is well known that much of the impairment of visibility in urban areas of the United States is the result of the scattering of light by airborne particles. Since evidence suggests that sulfates may be one of the most important contributors to visibility reduction by this means, an assessment of the benefits of

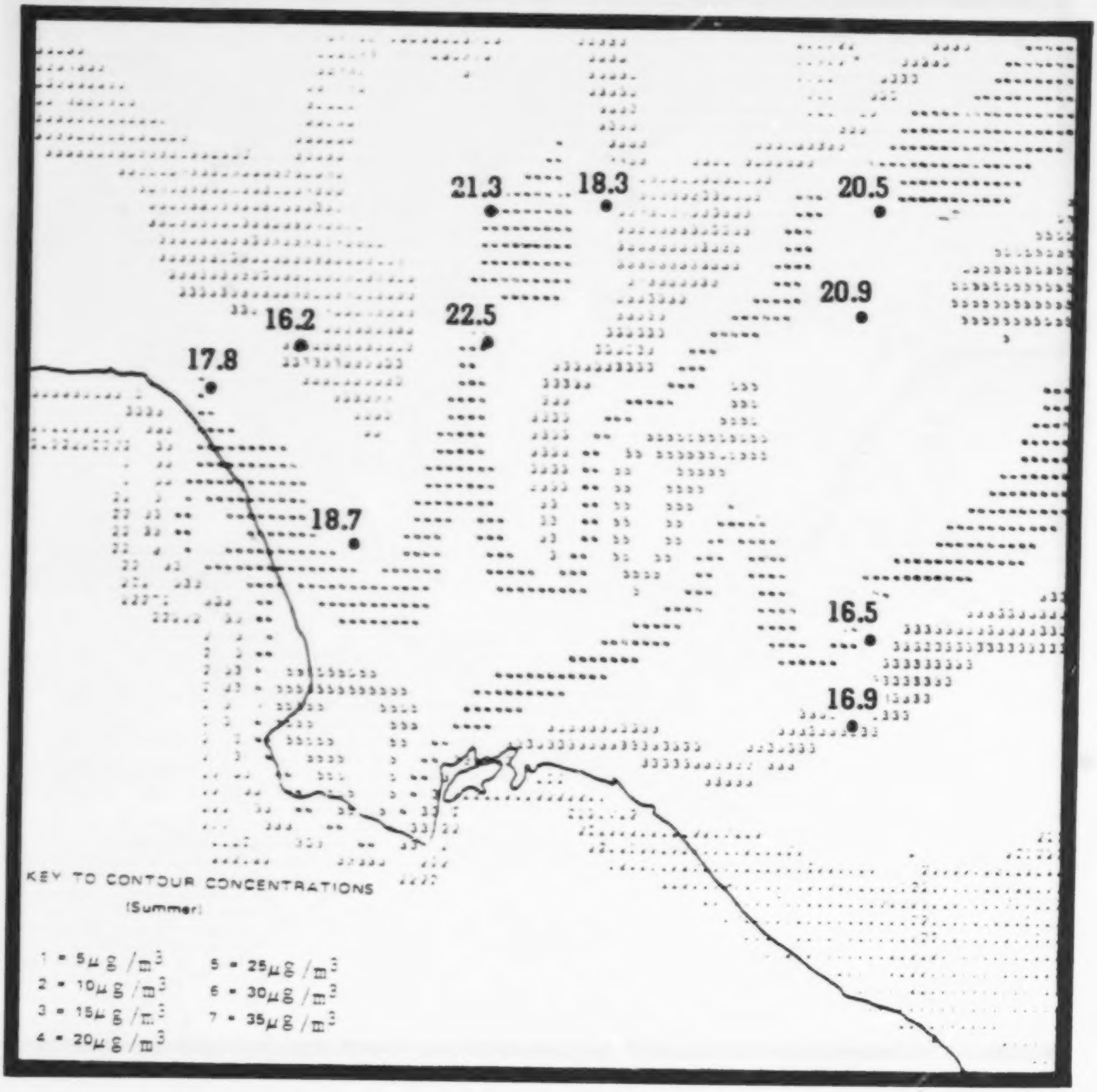

21.3
18.3
20.5
20.9
16.2
22.5
17.8
18.7
16.5
16.9
KEY TO CONTOUR CONCENTRATIONS
(Summer)
1 = 5μg/m³
2 = 10μg/m³
3 = 15μg/m³
4 = 20μg/m³
5 = 25μg/m³
6 = 30μg/m³
7 = 35μg/m³

Figure 5-8. Predicted and measured average $SO_4^{=}$ concentrations for summer, 1973, in the Los Angeles Basin. Contours indicate areas of approximately constant concentrations. Actual measured values are shown to one decimal place adjacent to monitoring sites. Numbers on controus indicate concentration of contour. All values are micrograms per cubic meter.

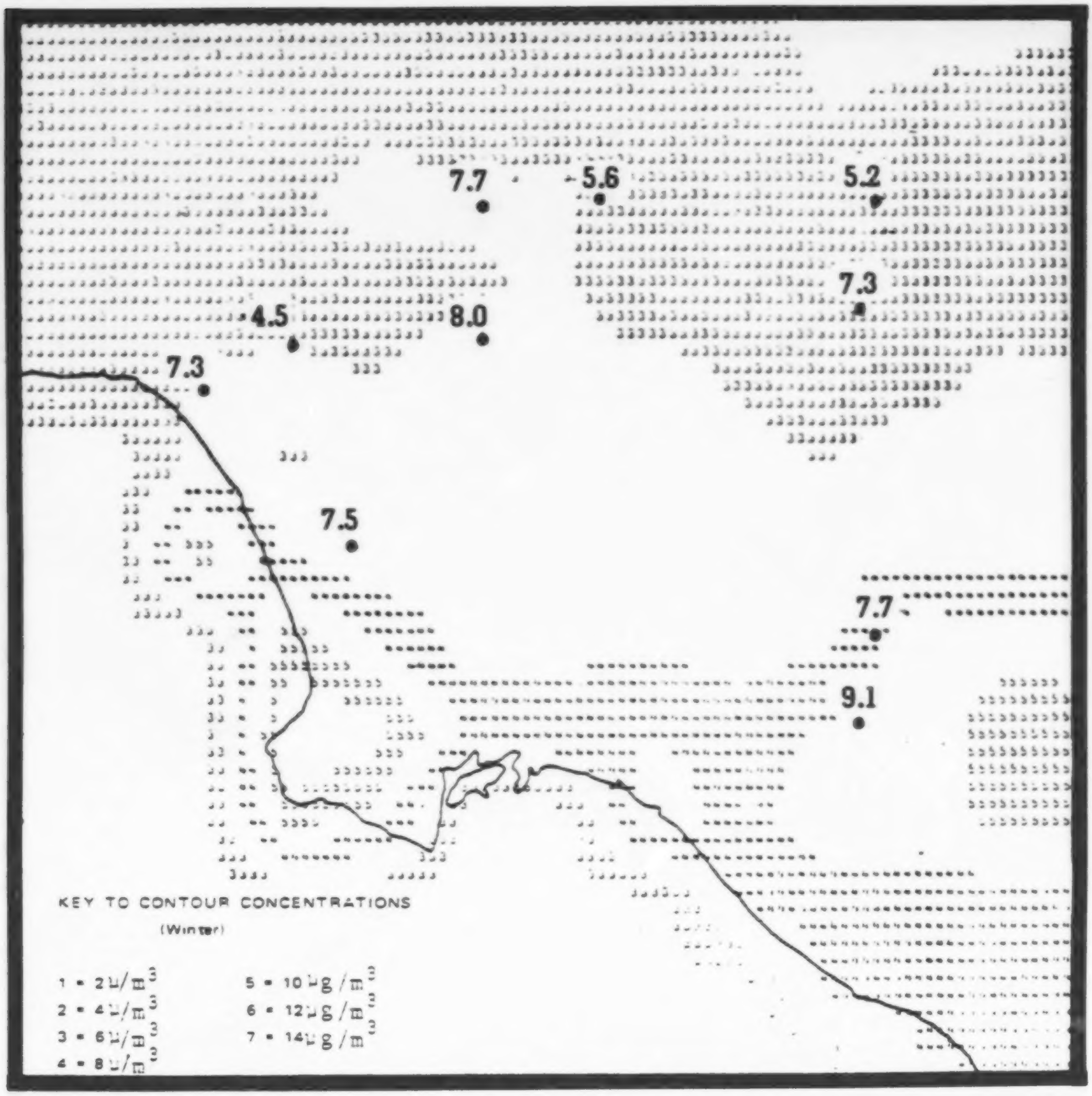

7.7
5.6
5.2
7.3
4.5
8.0
7.3
7.5
7.7
9.1
KEY TO CONTOUR CONCENTRATIONS
(Winter)
1 = 2µ/m³
2 = 4µ/m³
3 = 6µ/m³
4 = 8µ/m³
5 = 10µg/m³
6 = 12µg/m³
7 = 14µg/m³

Figure 5-9. Predicted and measured average $SO_4^=$ concentrations for winter, 1973, in the Los Angeles Basin. Contours indicate areas of approximately constant concentrations. Actual measured values are shown to one decimal place adjacent to monitoring sites. Numbers on contours indicate approximate concentration of contour. All values are micrograms per cubic meter.

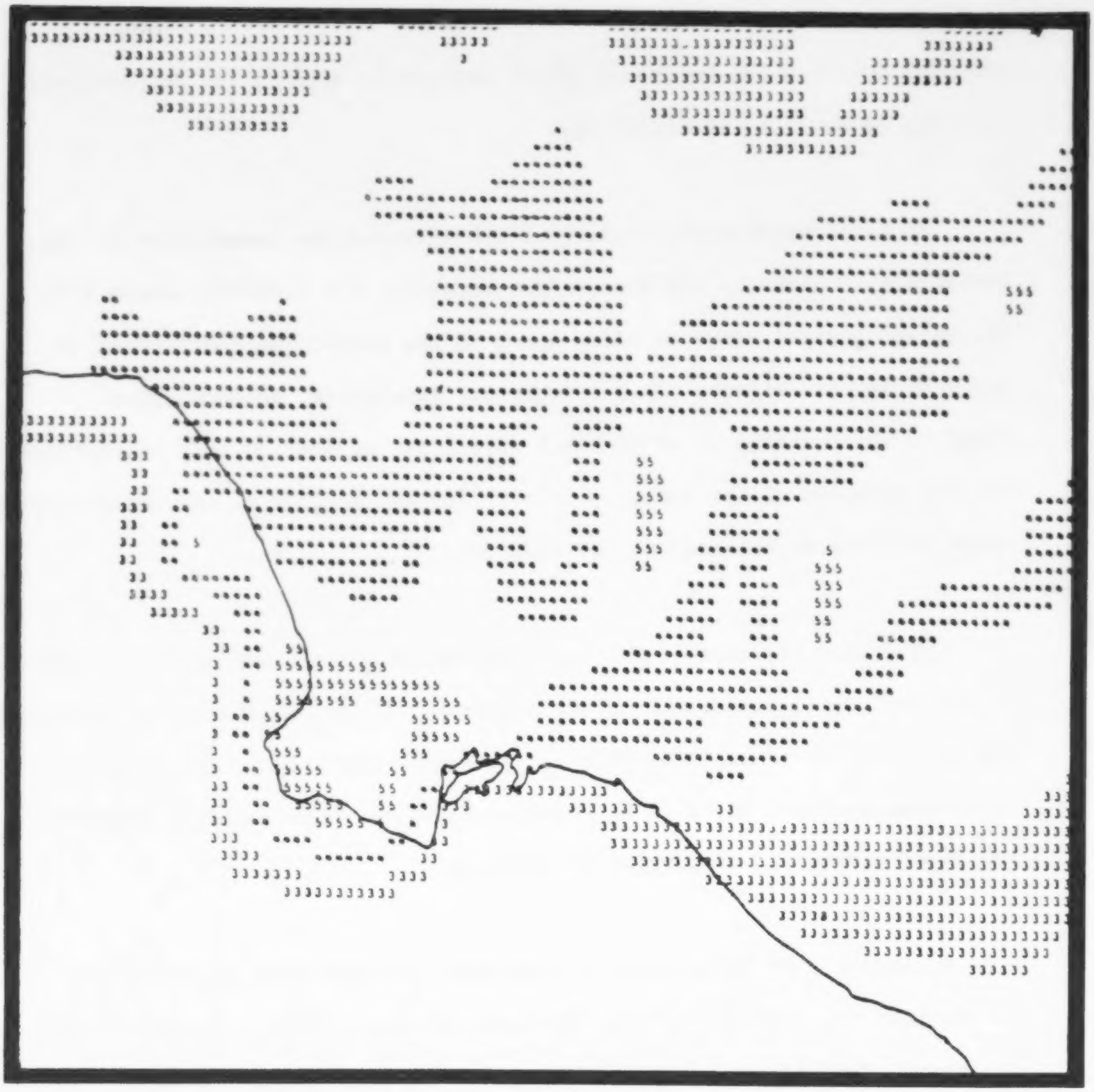


Figure 5-10. Total predicted annual average sulfate concentrations, 1980.

reducing sulfur oxide emissions should attempt to account for improvements that may be obtained in visibility.

The basic relationship between visibility and the composition of the atmosphere is given by the Koschmeider equation, $V = 0.0024/b$, where V is the meteorological range in miles and b is the extinction coefficient in units of inverse meters. In a homogeneous atmosphere, meteorological range is the distance at which black object can be seen against the horizon. For the purposes of the analysis, visibility was defined as the meteorological range as given by the Koschmeider equation.

The extinction coefficient b is the sum of the extinction coefficients of the various light-scattering and light-absorbing elements in the atmosphere. The contribution of sulfates to b is determined not only by the sulfate mass concentration, but also by relative humidity; high humidity enhances the light-scattering properties of sulfates.

An analysis of the effects of atmospheric composition on visibility in downtown Los Angeles has been performed by Cass (1978). In this study, long-term (1965-74) data accumulated by the Los Angeles Air Pollution control District were used to determine the parameters of a theoretically motivated form for the extinction coefficient b as a function of recorded relative humidity and levels of various atmospheric pollutants, including $SO_4^=$. The functional form of b was then used to predict the effect of a 50 percent and a

75 percent reduction in sulfate concentration on visibility; the historical data with reduced sulfate levels were used to calculate a reduced extinction coefficient b and, with the Koschmeider equation, the improved visibility V. The advantage of this method is that it preserves the correlation among various particulate levels and meteorological conditions. The output for each assumed sulfate reduction is not an average visibility, but a frequency distribution describing the percent of time that visibility is less than any given value.

For a given location, frequency distributions describing visibility tend to have the shape of a log-normal distribution. This is also true for the frequency distributions predicted by Cass. Thus, the results of Cass's analysis and the assumption of a log-normal frequency distribution for daily average visibility make it possible to express the parameters of the log-normal distribution describing visibility in downtown Los Angeles as a function of the fraction of atmospheric $SO_4^=$ removed.

For this analysis, it was necessary to estimate future rather than past visibilities and to perform this estimate for other areas in the basin in addition to downtown Los Angeles. Considerably more data are needed to determine exactly what variations occur in these frequency distributions from location to location and from year to year. For the purpose of this analysis, the simplifying assumption was made that the relative contributions of the various components to the extinction coefficient were approximately the same, regardless of location or year. With this assumption, the frequency distributions, given no additional controls on SO_x emissions, would be identical except for scaling factors based on differences in total particulate matter concentrations. Thus, estimates of the frequency distribution describing 1980 visibilities could be obtained at any location given only the total 1980 particulate level at that location relative to the level in downtown Los Angeles from 1965 to 1974 and given the fraction of the base case $SO_4^=$ that is removed from the atmosphere under the assumed control policy.

Figure 5-11 shows an example of the results obtained by using this approach. The figure shows the frequency distribution obtained for 1980 visibilities at downtown Los Angeles under the base case assumption and under the assumption of complete desulfurization of gasoline. At this location, desulfurization resulted in about a 10 percent improvement in average visibility. Table 5-6 summarizes the results for all cells by showing the average visibility obtained under each of the emission assumptions.

Table 5-6. AVERAGE VISIBILITY OVER THE LOS ANGELES BASIN UNDER EACH AUTOMOBILE SOx EMISSION ASSUMPTION (in miles)

Control	Degree of control Minimum	Moderate	Maximum
Reduce fuel sulfur	3.69	3.83	3.85
Reduce conversion	3.71	3.75	3.76
Trap $SO_4^=$	3.74	3.75	3.76

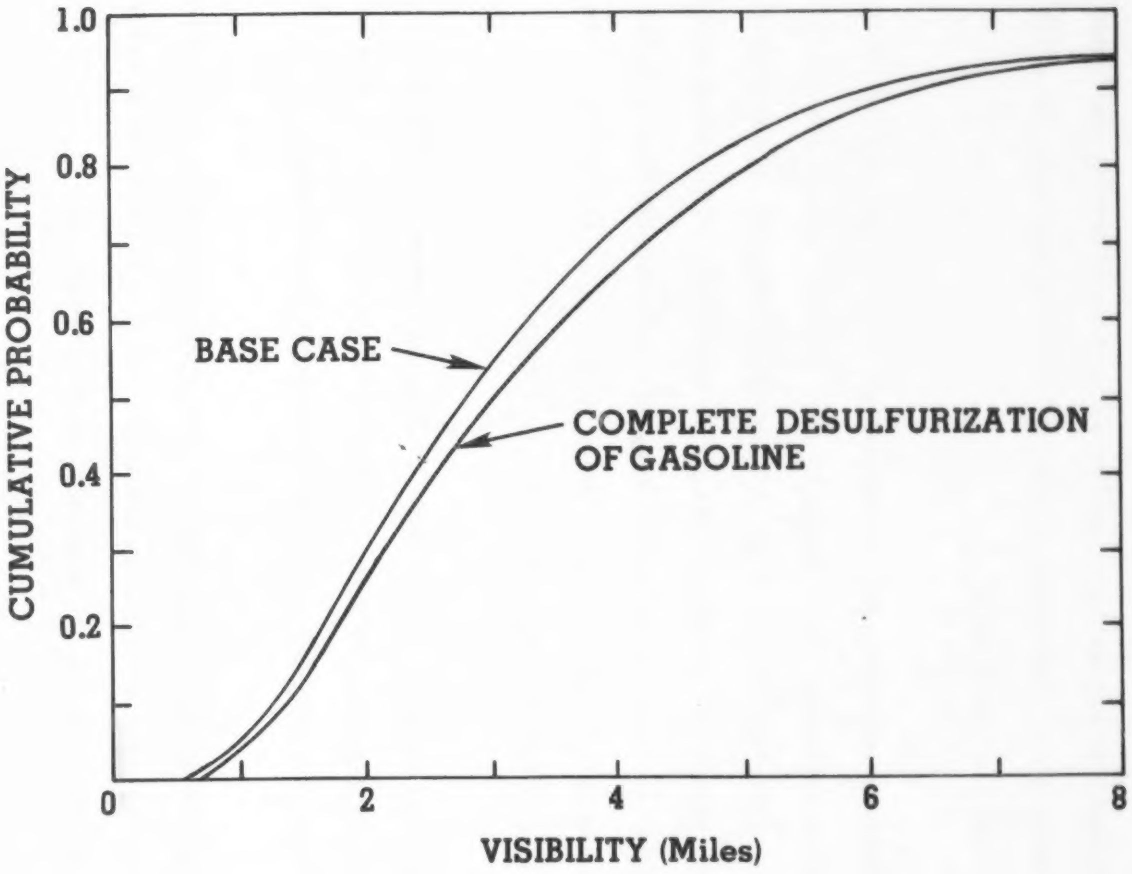


Figure 5-11. Probability that a given measurement of 1980 downtown Los Angeles visibility will be less than or equal to any distance under the base case emission assumptions and under complete desulfurization of gasoline.

People obviously prefer good to poor visibility, and it seems likely that at least some people would be willing to decrease their material standard of living in order to improve their local visibility. However, very few attempts at estimating a dollar value for visibility have been made, and there are no published studies of the economic value of visibility to the residents of Los Angeles.

To obtain a rough estimate of the dollar value of visibility improvements to Los Angeles residents, this analysis uses the preliminary results of a recent study performed in the city of Farmington, located in the scenic Four Corners region of New Mexico (Blank et al. 1978). This study employed a bidding game approach to determine how much each household or visitor in the area would be willing to pay to avoid reduction of visibility. The visibilities considered ranged from 25 to 75 miles.

Application of these results to Los Angeles, where visibility averages around 5 miles, required considerable extrapolation. Two alternative approaches were considered: the linear and logarithmic models.

For the linear model, the simplest interpretation of the Four Corners data is that the cost of reducing visibility from a value V_1 to a value V_2 is simply proportional to the magnitude of the reduction:

$$\begin{array}{l}\text{Social cost per person}\\ \text{per year of reducing}\\ \text{visibility from } V_1 \text{ to } V_2\end{array} = k_1 (V_1 - V_2)$$

With this interpretation, the Four Corners data yielded an average value for k_1 of \$0.70 per mile per person per year. This is computed from an average of values given in Blank et al. (1977) and assumes 2.57 persons per household. The result is in fairly good agreement with that of Randall et al. (1974).

The linear model predicts a social cost of a 1-mile reduction in visibility that is the same whether the reduction occurs where prevalent visibility is 75 miles or 5 miles. Another approach is to assume that the cost of visibility reduction is related to the percentage reduction in prevalent visibility. Therefore a logarithmic cost model was considered wherein the cost of reducing visibility from V_1 to V_2 is given by:

$$\begin{array}{l}\text{Social cost per person}\\ \text{per year of reducing}\\ \text{visibility from } V_1 \text{ to } V_2\end{array} = k^2 \log_{\text{base } 2} (V_1/V_2)$$

The factor k_2 may be interpreted as the social cost per person of a 50 percent reduction in visibility. With the Four Corners data, a value of \$24.50 per person per year was obtained for k_2.

Total visibility costs were computed for each of the geographic cells defined over the Los Angeles Basin by using both the linear and logarithmic cost models. The total visibility costs obtained by summing over cells are shown for each emission control assumption in Table 5-7. In all cases, the visibility costs were measured relative to the situation in which all $SO_4^=$ attributable to automobiles was removed from the atmosphere. Thus, the values shown in Table 5-7 can be interpreted as the social costs of visibility reduction attributable to automobile $SO_4^=$ under each emission control assumption.

Table 5-7. VISIBILITY COSTS ATTRIBUTABLE TO
AUTOMOBILE SOx EMISSIONS, 1980
(millions of dollars)

Control	Degree of control					
	Minimum		Moderate		Maximum	
	Linear model	Log model	Linear model	Log model	Linear model	Log model
Reduce sulfur	1.1	17.2	0.1	2.5	0	0
Reduce conversion	1.0	15.5	0.7	10.7	0.6	9.3
Trap $SO_4^=$	0.8	13.0	0.7	10.8	0.6	9.2

[a] Base case: $0.8 million (linear model); $13.0 million (log model).

Considerably more visibility costs were predicted under the log model than under the linear model. For a conservative estimate (large visibility costs), total social costs are computed assuming the log model.

5.2.1.4 Damage to vegetation--At this time, there are no reported instances of vegetational injury induced by sulfur compounds generated by mobile or line sources. Recently automobile-generated sulfate aerosol was considered to be a potential threat to vegetation. However, based on the ambient air quality data and preliminary information on vegetational dose-response relationships to submicron H_2SO_4 aerosols, sulfate aerosols currently are not considered to be directly injurious to plants in the vicinity of line sources.

Benedict et al. (1973) estimated the total losses attributed to sulfur oxides singly and combined with other pollutants. According to their estimates for EPA Region IX (Table 5-8), total losses exceed $80 million. A best-guess estimate of the damage caused by mobile sources in the Los Angeles Basin is $1 million.

Table 5-8. ESTIMATED DAMAGE TO VEGETATION IN EPA REGION IX (dollars)

Plants	Value in polluted areas	Losses due to sulfur oxides	Total loss due to ozone, SOx, and fluorides
Citrus	162,650.3	0.3	6,224.5
Field crops	722,905.1	1,442.0	7,549.9
Seed crops	45,610.7	1.6	444.1
Fruits and nuts	460,158.1	1.4	8,965.2
Vegetables	633,731.0	220.0	10,205.1
Nursery and forest	155,077.2	0.4	18,905.6
crop total	2,180,132.4	1,665.7	52,274.4
Forestry	419,062.2	993.1	20,001.8
Highways	22,349.6	33.5	2,629.4
Parks	38,118.3	40.4	2,319.4
Residential	39,309.6	131.1	3,337.8
Urban uses	2,549.2	16.7	127.3
Rural uses	27,436.6	142.3	767.5
Ornamentals total	548,825.5	1,357.1	
Total all plants	2,748,957.9	3,022.8	81,457.6

5.2.1.5 Damage to nonbiological materials--Appendix B contains estimates of materials damage caused by sulfur-bearing compounds emitted from both stationary and mobile sources. The social costs and benefits of controlling the mobile emissions are determined by factoring the total nationwide damage estimates by the percent of mobile source emissions. Less than $1 million in nonbiological materials damage is attributed to mobile source sulfur emissions in the Los angeles Basin.

5.2.1.6 Benefits analysis--The estimated social welfare costs and benefits of automotive sulfur oxide emission controls in the Los Angeles Basin for the year 1980 are listed in Table 5-9. The social costs of reduced visibility are based in this section on a 50 percent reduction in sulfur oxide emissions from stationary sources. The previous costs of visibility reduction assumed emissions from stationary sources of 743 tons per day. The projected annual average sulfate concentrations under the 50 percent reduction assumption were between 12 and 16 $\mu g/m^3$.

The social costs of biological and nonbiological materials damage were given the nominal value of $2 million. This value was treated under the assumption used for estimating visibility costs, i.e., as a function of the fraction of atmospheric sulfate removed. All benefits listed in the table are defined as a decrease in social costs.

Table 5-9.

SOCIAL WELFARE COSTS AND BENEFITS OF AUTOMOBILE SULFUR OXIDE EMISSION CONTROLS ASSUMING LOWER STATIONARY SOURCE EMISSIONS FOR THE LOS ANGELES BASIN, 1980 (millions of dollars)[a]

	Degree of control								
Control	Visibility	Minimum Vegetation/non-biological material	Benefits	Visibility	Moderate Vegetation/non-biological material	Benefits	Visibility	Maximum Vegetation/non-biological material	Benefits
Reduce fuel sulfur	26.5	2.8	-8.1	3.9	0.4	16.9	0	0	21.2
Reduce Conversion	23.9	2.5	-5.2	16.5	1.7	3.0	14.5	1.5	5.2
Trap $SO_4^{=}$ emissions	19.2	2.0	0	16.8	1.8	2.6	14.3	1.5	5.4

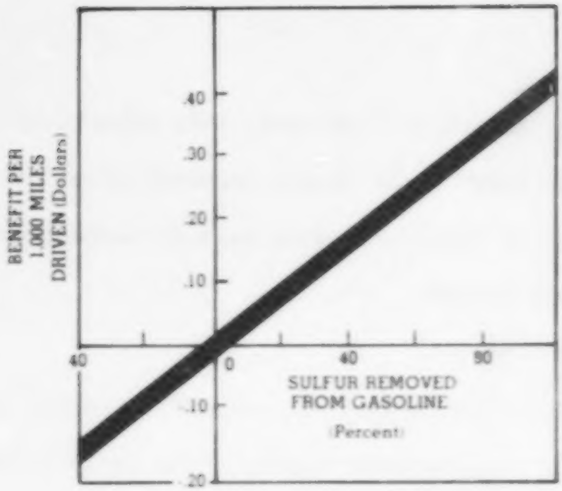
[a] Costs attributable to automobile sulfur oxide emissions.

5.2.1.7 Cost/benefit comparison--Figure 5-12 shows curves obtained from the results of the benefit analysis when benefits are plotted in terms of dollars per thousand vehicle miles traveled. Because each curve is based on only three or four points and because the exact values of these points are highly uncertain, these curves must be interpreted with extreme caution. Benefits accounted for in the analysis were improved visibility and reduced damage to biological and nonbiological material. To facilitate the comparison of benefits with the costs of controls, benefit estimates were expressed in equivalent dollar values.

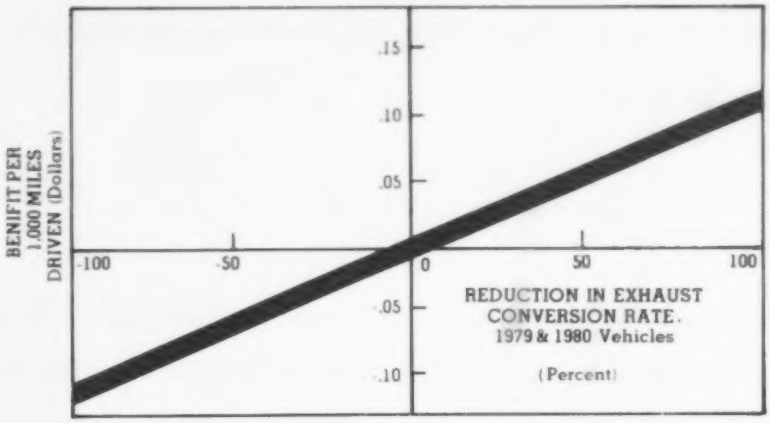
Estimated total benefits were found to be relatively insensitive to the level of assumed 1980 stationary source emissions: about $17 million for desulfurization of gasoline (to 0.01 percent sulfur) and about $3 million for controls that reduce sulfate emissions only (30 percent reduction in sulfate emissions for 1979 and 1980 vehicles).

Unfortunately, little information concerning the costs and effectiveness of devices for controlling SO_x emissions from automobiles was available at the time of this study. However, the use of the results of the benefit analysis for performing cost/benefit comparisons are illustrated using preliminary cost estimates for desulfurization of gasoline.

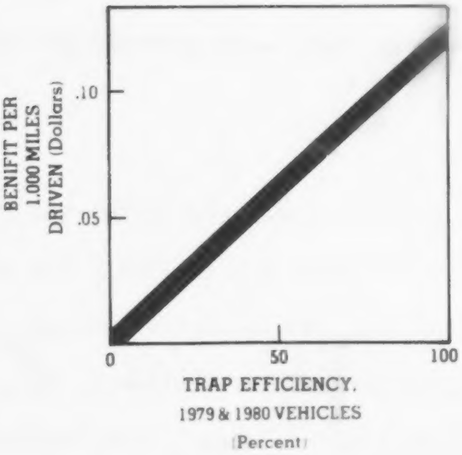
From Kittrell and Short (1978), West Coast manufacturing costs for desulfurizing gas to 100 ppm (0.01 percent) were estimated to be roughly $.02 per gallon. Using base case assumptions for vehicle fuel mileage, an average fuel economy for 1980 on-the-road vehicles of 16.6 miles per gallon was computed. Assuming an



(a) DESULFURIZATION



(b) IMPROVED CATALYST



(c) TRAP

Figure 5-12. Benefit per 1000 miles driven versus degree of control.

energy penalty of desulfurization of 1 percent, this results in an estimated incremental cost to the consumer of $1.22 per thousand miles driven. From Figure 5-11a, the benefits of desulfurization to 0.01 percent were estimated at $0.34 per thousand miles driven.

The costs and benefits from controlling automotive sulfur emissions are the most favorable for fuel desulfurization. The costs of control cannot, realistically, be applied to other regions of the United States. In addition to the uncertainties associated with this analysis, the Los Angeles Basin is considered as a worst case and should not be compared with other metropolitan areas.

5.2.2 Kansas City, Mo., Study

The metropolitan area of Kansas City, Mo., was selected as a typical large urban area in the United States. This area also provides data from a recent diffusion modeling study prepared by PEDCo for the U.S. Environmental Protection Agency (1978) on particulate emissions from diesel-powered vehicles. The data derived in that study were modified for this exposure assessment to sulfates.

Base year emissions from mobile sources are calculated from 1974 vehicle exhaust information and are projected to 1985. The vehicle miles traveled (VMT) are estimated for six vehicle/engine classes. The percent of VMT by vehicle type, the vehicle type distribution by age, and the rate of diesel-powered vehicle introduction are used in conjunction with the Kansas City traffic distribution and growth rate. The projected motor vehicle sulfate exhaust emissions are noted in Table 5-10. The emission rates are based on diesel vehicle introduction rates (Table 5-11) and fraction of VMT by mobile source catagory (Table 5-12).

The projected annual average concentrations of sulfates from diesel exhaust are projected through 1985 in Table 5-13. The ambient concentrations from the diesel source incremental contribution were derived from the Air Quality Display Model. Under a maximum penetration of diesel-powered vehicles, a maximum sulfate concentration of 0.02 $\mu g/m^3$ from diesels is forecasted. This small increment would not hold true for sulfur dioxide, which, because of time limitations, was not modeled for this area.

5.3 CONCLUSION

Two scenarios have been presented to estimate exposure levels to primary sulfur emissions from mobile sources. The social welfare costs and benefits of reducing these emissions were explored for an atypical area, the Los Angeles Basin. The emission/modeling studies show that sulfuric acid emissions from catalyst-equipped automobiles disperse rapidly into the ambient air. The automotive increment to the total ambient sulfate concentrations averages 2 $\mu g/m^3$. Levels as high as 8 $\mu g/m^3$ were found at an elevation of 1.5 feet both above and near the roadway. Concentrations occurring inside the automobiles ranged from 1 to 4 $\mu g/m^3$.

Table 5-10. PROJECTED MOTOR VEHICLE EXHAUST EMISSIONS (SULFATES)
(ton/yr)

Vehicle category	1974	1981		1983		1985	
		Best est.	Max.	Best est.	Max.	Best est.	Max.
Gasoline-powered							
Light-duty vehicles	13.0	3.5	3.4	2.0	1.5	1.3	1.2
Light-duty trucks	1.7	0.5	0.5	0.4	0.4	0.2	0.2
Heavy-duty trucks	2.0	2.2	2.1	2.2	1.9	2.2	1.5
Subtotal	16.7	6.2	5.9	4.6	3.8	3.7	2.9
Diesel-powered							
Light-duty vehicles	0.1	0.5	0.5	1.2	2.9	1.9	4.6
Light-duty trucks	<0.1	<0.1	0.1	0.1	0.4	0.2	0.6
Heavy-duty trucks	33.3	3.8	4.1	4.1	4.8	4.2	5.8
Subtotal	3.5	4.4	4.7	5.7	8.1	6.3	11.0
Total	20.2	10.6	10.6	10.3	11.9	10.0	13.9

[a] "Best est." and "max." refer to diesel introduction rate assumptions.

Table 5-11. DIESEL VEHICLE INTRODUCTION RATES

	Share of new sales by model year			
	Light-duty vehicles and trucks, %		Heavy-duty trucks, %	
	Best est.	Max.	Best est.	Max.
1975	0.5	0.5	28.0	28.0
1976	0.5	0.5	28.0	28.0
1977	0.5	0.5	28.0	28.0
1978	0.5	0.5	30.0	35.0
1979	2.0	5.0	31.0	36.0
1980	4.0	10.0	31.0	38.0
1981	6.0	15.0	31.0	40.0
1982	8.0	20.0	31.0	48.0
1983	10.0	25.0	31.0	57.0
1984	10.0	25.0	31.0	67.0
1985	10.0	25.0	33.0	78.0

Table 5-12. FRACTION OF URBAN VMT BY MOBILE SOURCE CATEGORY IN PROJECTION YEARS

	Fraction of urban VMT						
	1974	1981		1983		1985	
Mobile source category		Best est.	Max.	Best est.	Max.	Best est.	Max.
Light-duty gasoline vehicles	0.826	0.815	0.796	0.798	0.750	0.779	0.704
Light-duty gasoline trucks	0.107	0.106	0.104	0.104	0.098	0.102	0.093
Heavy-duty gasoline trucks	0.036	0.035	0.033	0.034	0.029	0.034	0.023
Light-duty diesel vehicles	0.004	0.015	0.034	0.032	0.080	0.051	0.126
Light-duty diesel trucks	0.001	0.002	0.004	0.004	0.010	0.006	0.015
Heavy-duty diesel trucks	0.026	0.027	0.029	0.028	0.033	0.028	0.039

Table 5-13. PROJECTED REGIONAL ANNUAL AVERAGE CONCENTRATIONS FOR SULFATES FROM DIESEL EXHAUST FOR TEST CITY
($\mu g/m^3$)

Year	Diesel growth case	
	Best ext.	Max. growth
1974	0.009	
1981	0.009	0.011
1983	0.011	0.017
1985	0.013	0.023

The automotive sulfate increment to the total stationary source sulfate emissions was also modeled for the Los Angeles Basin. The benefits are listed in summary Table 5-14. Benefits are defined as a decrease in social costs.

TABLE 5-14. ESTIMATED SOCIAL WELFARE BENEFITS OF MORE STRINGENT AUTOMATIVE SOx EMISSIONS CONTROLS, 1980
(millions of dollars)

	Degree of control		
Control	Minimum	Moderate	Maximum
Reduce fuel sulfur	-8.1	16.9	21.2
Reduce conversion	-5.2	3.0	5.2
Trap sulfate emissions	0	2.6	5.4

The costs of reducing mobile source sulfur emissions were estimated for fuel desulfurization at the moderate control level. In the Los Angeles Basin, the costs to the consumer were estimated at $1.22 per thousand miles driven. The benefits gained from this control measure are based primarily on an increase in visibility. Under the moderate fuel desulfurization scenario, a benefit of approximately $17 million was estimated for about a 0.1-mile increase in visibility.

One conclusion that can be drawn from the emission/modeling data in this chapter is that although sulfur emissions from mobile sources account for only about 2 percent of the total anthropogenic sulfur emissions nationwide, the percent of mobile source sulfate emissions is much higher than that of stationary source sulfate emissions.

Finally, the exposure assessments modeled for sulfate emissions from mobile sources should help future estimates for health effects once health effect levels for sulfates are established. The worst-case exposure estimates for sulfur dioxide indicate that the increase in sulfur dioxide emissions from an increase in diesel-powered vehicles is not likely to affect adversely the public health.

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6. SUMMARY AND CONCLUSIONS

An estimated 30 million tons of sulfur oxides are emitted annually in the United States. Because transportation vehicles account for only about 2 percent of this emission, their contribution to sulfur pollution was at one time considered relatively minor. This outlook changed when it was observed that the oxidation catalyst, designed to control other pollutants from automobiles, converted the sulfur dioxide (SO_2) in exhaust to sulfuric acid (H_2SO_4), a much more toxic compound.

Initial estimates showed that high amounts of H_2SO_4 were emitted from catalyst-equipped vehicles, more than twice the actual 10-15 mg/mile rate determined subsequently. This preliminary finding led to the establishment of the U.S. Environmental Protection Agency's Catalyst Research Program, which assessed the environmental impact of emissions from oxidation catalyst-equipped vehicles. This program and other related research projects identified the types of sulfur compounds emitted from mobile sources. In addition to sulfuric acid (which forms sulfate salts) and sulfur dioxide, hydrogen sulfide and carbonyl sulfide were found, but in trace amounts and only after control system failure. It was determined that high amounts of sulfates and sulfur dioxide were also produced by light-duty diesel vehicles. Consequently, the effects on the public health and welfare of these sulfur-bearing compounds were reviewed for this special report to the U.S. Congress under Section 403(g) of the 1977 Amendments to the Clean Air Act.

Toxicological studies show that sulfuric acid is the most irritating of the atmospheric sulfate ($SO_4^=$) compounds. As little as 70 $\mu g/m^3$ H_2SO_4

can narrow the larger air passages (bronchi) of the lungs in animals, an effect which is reflected by an increase in airway resistance. To produce the same degree of bronchoconstriction, a more than threefold higher concentration of SO_2 is required. One reason for the relatively high toxicity of H_2SO_4 is the ability of sulfate particles to reach the deep lungs more readily than SO_2, which tends to be absorbed in the moist mucous membranes of the upper airways. When SO_2 is combined with small particulate matter, it enters more deeply into the lung and exerts stronger effects.

Exposure to H_2SO_4 at concentrations of 350-5000 $\mu g/m^3$ for 5-15 min has been shown to affect respiration rates in healthy adults. The absence of effects at lower concentrations, even in sensitive subjects such as asthmatics, is apparently due to the presence of ammonia in the mouth, which neutralizes the acid aerosol at these lower test concentrations.

For several years following publication of the criteria documents for SO_x and particles, most of EPA's air pollution studies on sulfates and SO_2 were conducted under a large-scale program known as CHESS. The research data indicated that acute responses, specifically aggravation of asthma and symptoms in the elderly, were associated with 24-hr exposures to 10-15 $\mu g/m^3$ sulfate and 200-300 $\mu g/m^3$ SO_2. Chronic effects were observed at 200-400 $\mu g/m^3$ SO_2, 7-20 $\mu g/m^3$ $SO_4^=$, and 60-165 $\mu g/m^3$ TSP (total suspended particles).

Although the CHESS studies had several flaws, including problems in sorting out variables that were causally related to the observed effects, they pointed up the effects on morbidity and mortality of sulfur oxides and associated particulate matter. Additional research is needed to establish dose-response relationships for sulfates before public health effects can be quantified.

Of all airborne sulfur pollutants, sulfur dioxide is the most damaging to vegetation. This damage ranges from discoloration and leaf shedding to reduced growth rates. Vegetational injury attributed to mobile source sulfur emissions can be estimated roughly by factoring the damage from all SO_x emissions by the percent of mobile source emissions. The actual loss from damage caused by mobile sources is difficult to determine by current field assessment methods. One problem is the inability to sort out the effects of SO_2 alone from those of SO_2 in combination with other pollutants.

Assessment of damage to nonbiological materials is plagued by similar problems. There have been no studies to isolate the relative contributions of mobile versus nonmobile sources. The effects of total emissions from all sources, however, can be analyzed to estimate the materials damage from mobile emissions. Studies show that sulfur oxides, salts, and acids can etch metal surfaces such as galvanized coatings; discolor painted surfaces, fabric, and building materials; and leach carbonate building stone. Nevertheless, it is difficult to distinguish the damage caused by sulfur compounds from that caused by other pollutants and natural processes.

The effects of sulfur compounds on climate cannot yet be determined. Although sulfate particles can absorb and scatter energy from the sun and, possibly, affect precipitation, no relationship between climatic alterations and pollutant concentrations has been established. Effects on visibility, however, have been noted.

Recent studies suggest that sulfates may be a principal cause of visibility reduction in certain areas of the United States. For example, regression models based on variations in pollutant concentration and visibility indicate that sulfates may account for 50 percent of the total visibility reduction in the East and along the West Coast.

Various alternatives to control sulfur emissions from mobile sources have been investigated. Of these, fuel desulfurization and the three-way catalyst (TWC) are considered among the most promising. Desulfurization of gasolines and diesel and jet fuels is now both economically and technologically possible. Both sulfuric acid and sulfur dioxide can be reduced effectively by this control measure. However, the cost is high, $1.22 per 1000 miles driven. The second control alternative, the TWC, does not affect total sulfur emissions but does reduce sulfuric acid to less than half the amount emitted by the oxidation catalyst. The TWC is currently used in some domestic and foreign model vehicles to control federally regulated emissions and provides the additional cost-free benefit of controlling sulfuric acid.

The lack of adequate dose-response data precludes an analysis of the health benefits to be gained using these control alternatives to reduce ambient sulfate levels. Measurements of sulfate levels on busy urban freeways have shown that the highest concentrations occurring inside the vehicles averaged 1-4 $\mu g/m^3$, while typical congested highway exposures average less than 2 $\mu g/m^3$. Probability studies indicate that even under conditions most conducive to high roadway sulfate levels, there is less than a 1 percent chance of a several-minute exposure to more than 6 $\mu g/m^3$. These various studies show that because automotive sulfuric acid emissions disperse rapidly into the ambient air, high localized concentrations are not likely to occur.

The damage to vegetation and nonbiological materials by mobile source sulfur emissions can be quantified in economic terms more readily than the damage to visibility. Vegetation and nonbiological materials damage amounts to less than $35 million nationwide. An assessment of the costs associated with visibility reduction is based primarily on subjective values and, consequently, has a greater margin of error. Moreover, shifts in the stationary source emission of sulfur oxides would have significantly greater impact on the mobile source contribution to visibility reduction than to vegetation and nonbiological materials damage.

Current exposure estimates suggest that major adverse health and welfare effects from mobile source emissions of sulfur-bearing compounds are unlikely. Consequently, specific control of the mobile source emissions of sulfur-bearing compounds is not recommended at present.

APPENDIX A. HEALTH EFFECTS

A.1 INTRODUCTION

After the 1930 air pollution episode in the Meuse Valley, Belgium, a prediction was made that if a similar episode hit London, England, over 3000 people would die. In 1952, this prediction came true when a heavy fog of pollution blanketed London and left 4000 dead and many more thousands ill.

Fortunately, disasters such as these occurred rarely and will probably never occur again. With the exception of the air pollution episode in Donora, Pennsylvania, which caused 20 deaths, and in New York City, pollution episodes in the United States have been linked more with morbidity factors than mortality.

To provide additional information on air pollution and human health, many investigators have attempted to delineate the cause-effect relationships of exposure to sulfur-bearing compounds. This relationship is complex; nevertheless, there is sufficient information to conclude that sulfur-bearing compounds are toxic. Their toxicity depends on the particular sulfur compound, the concentration and time of exposure, and other covariants such as additional pollutants or preexisting disease. This type of information is derived from four major experimental approaches: laboratory studies *in vitro*, animal toxicology studies *in vivo*, human clinical studies, and epidemiological studies.

A.2 IN VITRO

A.2.1 Introduction

In vitro studies have disclosed various toxic effects from sulfur dioxide and sulfate inhalation. Sulfur dioxide hydrates fairly rapidly in the upper airways to form several ions. One of these, a bisulfite ion, reacts readily with cellular genetic material, with serum proteins, and probably with cellular proteins. Its effects on catalytic proteins, such as enzymes, have varied from inhibition and stimulation to no observable effect.

Bisulfite degrades both DNA and RNA significantly--modifications which can result in mutations. The mutagenic specificity of bisulfite has been demonstrated in bacteria, viruses, yeast, and cultured mammalian cells. This mutagenic action, however, has not been demonstrated in *in vivo* studies. Aside from the general lack of bisulfite mutagenesis studies *in vivo*, several protective mechanisms could increase its chemical transformation or could sequester it until urinal excretion. The amount of bisulfite and degree of ionization determine its ability to enter a cell and affect genetic material. In addition, bisulfite could be oxidized in the body to the less toxic sulfate ion.

Whether SO_2 inhalation at present atmospheric concentration is likely to lead to greater mutations in man cannot be determined from the available data. Mathematical models could be developed to describe SO_2 absorption and the reactions of SO_2 leading to mutations in an effort to predict the hazard from present levels. Direct experiments with animals are not likely to be fruitful, since the mutation rate, if one exists at all, is probably near the limit of detection by present methods. This is not to say that a potential hazard does not exist, only that proper experiments have not been devised to test this question.

On the other hand, other effects seen in *in vitro* and *in vivo* studies do overlap. Sulfate aerosols (and sulfur dioxide) produce bronchoconstriction in animals and man. This constriction appears to arise from the release of histamine by sulfate salts, probably through an ionic mechanism within the cell. In addition, the potency of different sulfate salts to promote bronchoconstriction or to release histamine suggests that different chemical compositions of atmospheric aerosols exert different effects on health. For instance, the more potent sulfate salts are those most likely found in the smaller (<3 μm) particles. The acidity of the aerosol is also important. Solutions more acidic or basic than the physiological pH increase the rate of sulfate removal from the upper airways. Finally, the chronic effects of sulfate inhalation are likely to involve cell-mediated inflammation, since sulfate promotes the release of the highly reactive free-radical species of oxygen, the superoxide radical ion.

A.2.2 SO_2/HSO_3^-

A.2.2.1 *Respiratory tract transport and absorption*--Sulfur dioxide is a highly water-soluble gas which hydrates rapidly in solution to form sulfurous acid, bisulfite, and sulfite ions:

$$SO_2 + H_2O \rightarrow [H_2SO_3] \rightarrow HSO_3^- + H^+ \rightarrow SO_3^= + 2H^+$$

The extent of ionization and relative contribution of each species depends on the pH and temperature of the solution. However, the relative significance and even existence of sulfurous acid in this equilibrium scheme is questionable (Cotton and Wilkinson 1972). The chemical state of sulfur dioxide, the prime determinant of the compound's toxicity, is important only in affecting the compound's ability to equilibrate with a body compartment. Therefore, the terms bisulfite and sulfur dioxide designate the same reactive species regardless of the chemical used to prepare an experimental solution.

Sulfur dioxide and sulfite salts enter the body primarily through inhalation of atmospheric sulfur dioxide and ingestion of sulfite salts found in foods, beverages, and drugs. In a model using an artificial tracheobronchial system lined with a simulated airway secretion (bovine serum albumin [BSA] dissolved in saline), SO_2 was absorbed primarily in the upper third of the simulated airway. Only a small fraction of the initial SO_2 reached that part simulating the alveolar and respiratory bronchial region, while none of it reached the terminal traps.

These results support known _in vivo_ effects, that is, that the rapid increase in airway resistance and excess mucus production associated with acute exposure to high concentrations of sulfur dioxide are due in part to the absorption by the upper airways. However, these data suggest that the gas will be readily hydrated in the tracheobronchial fluid and will exist predominantly as bisulfite, an ionic species which crosses cell membranes very slowly. Subsequently, the bisulfite may be carried up the bronchial tree by mucociliary transport and be ingested. It could then enter the stomach and exist there as sulfurous acid, which could be absorbed by way of the intestinal tract to the circulatory system and then carried in the blood to nonpulmonary sites. Thus, much of inhaled sulfur dioxide may become ingested bisulfite. The BSA/saline fluid used in this model, however, may not accurately reflect the actual biological system. Although albumin is often found in airway mucus, it is not the primary functional component of mucus. A greater portion of SO_2 may be sequestered effectively in the upper respiratory tract than is reflected in the BSA/saline model system (Gause and Barker 1978).

Sulfur dioxide in combination with an aerosol of sodium chloride (NaCl) was deposited in the more distal parts of the simulated lung than was SO_2. The exact physical-chemical mechanism for this deposition is not clear, but it appears that aerosol combinations with SO_2 could lead to altered effects, since hydration and removal in the upper airways would be circumvented and since chemical interaction of SO_2 and the particle could create a potentially reactive species.

Another inhalation mechanism involves the adsorption of SO_2/HSO_3^- on particles in the respirable size range. Since these particles would penetrate into the deep lung, SO_2 would be available to all lung cell types, such as alveolar macrophages, and to fluids of the alveolar region (Novakov et al. 1972).

A.2.2.2 Cellular effects--Alterations in lymphocytes induced by SO_2/HSO_3^- may explain the altered immunologic responses seen after in vivo inhalation of sulfur dioxide.

Schneider and Calkins (1970) isolated lymphocytes in a cell culture medium containing phytohemagglutinin (PHA), which induces blastogenesis and mitosis. After exposure to 500 cc of 5.7 ppm sulfur dioxide at varying time intervals, there was reduced cell enlargement, reduced DNA synthesis, a lower mitotic index, and clumping of chromosomes. All of these effects were most noticeable in the young cultures in which cells were preparing for DNA synthesis before mitosis. The results suggest that SO_2 induces damage to the genetic material of the lymphocyte.

Evidence for altered membrane function of human lymphocytes has been presented by Gause and Rowlands (1975), who found that 10^{-4} M sulfur dioxide or bisulfite caused aggregation, internalization, and increased rate of turnover of cellular membrane proteins. Alteration of membrane protein on lymphocytes may result in immunological aberrations _in vivo_, also affecting antigen recognition. If alteration of the nuclear material occurs as well, the composition and conformation of surface proteins may be changed.

The macrophage populations of a variety of species have also been studied as model systems for the action of sulfur dioxide _in vivo_, but _in vitro_ studies in this area are lacking. Because these cells are scavengers of foreign materials in the body and are important components of the host defense system, their alteration by pollutants may have significant systemic or localized effects.

Bourbon et al. (1973) determined the LD_{50} (dose which destroys 50 percent of the organisms studied) for rat alveolar macrophages to be a 1-hr exposure of approximately 10 mg/m^3 SO_2. An assay of the activity of the

Krebs cycle enzymes--lactic, maleic, isocitric, and succinic dehydrogenases--was also performed. A great increase in enzyme activity, and therefore metabolic rate, was observed in the macrophages which survived SO_2 exposure.

Gause et al. (1977) extended this work to include both *in vitro* exposure of baboon alveolar macrophages and *in vivo* exposure/*in vitro* assay of rat alveolar macrophages. They noted that macrophage ATPase activity (ATP utilization) was increased by both *in vivo* and *in vitro* exposure to SO_2. In addition, the levels of lysozyme activity were also increased by *in vivo* exposure.

Relatively few studies have focused on the effects of sulfur on blood and blood vessels. However, its effects on blood platelet aggregation and blood vessel contractility have been studied in isolated systems.

Kukugawa and Iizuka (1972) have shown that 10^{-3} to 10^{-2} M bisulfite inhibits the ADP- and collagen-induced aggregation of platelets, major components of the blood coagulating system. The inhibition was both direct and reversible, and it occurred in whole blood as well as in platelet-rich plasma. The mechanism of action and toxicological importance of these observations are unclear, but transient inhibition of platelet aggregation is probably not destructive.

Laszt and Schaad (1974) investigated the effect of bisulfite on the peripheral and pulmonary circulation by superfusing rings of peripheral blood vessels of the cow with solutions of 6 mM bisulfite with and without electrical stimulation. A gradual loss of tension and contractility of the carotid artery was observed, but the effect was reversible. In contrast, the pulmonary artery tone increased after bisulfite, whereas the pulmonary vein tone decreased. The net effect of inhaled SO_2 on vascular smooth muscle is not clear from these studies. Nevertheless, the constriction of bronchial smooth muscle upon SO_2 inhalation suggests the possibility of a direct action of SO_2 and bisulfite on smooth muscles.

A.2.2.3 *Biochemistry of SO_2/HSO_3^-*--Sulfur dioxide reacts readily with all major classes of biomolecules. The reactivity of this agent with nucleic acids, proteins, lipids, and other biological components has been repeatedly demonstrated *in vitro*. The extrapolation of the implications of these isolated reactions to human toxicity, however, remains a point of controversy.

A.2.2.3.1 *Reactions with DNA and RNA in relation to mutagenicity*. The reactivity of bisulfite with nucleic acids and mutagenesis induced by bisulfite has been reviewed by Shapiro (1977) and by Fishbein (1976). The reactions involved are varied, but the *in vitro* conditions under which they occur are sufficient to account for many of the mutagenic effects observed as well as for such functions as chromosome clumping observed by Schneider and Calkins (1970). The various mechanisms of reaction of bisulfite with nucleic acids will be considered first.

A specific deamination of cytosine to uracil in single-stranded, but not double-stranded, DNA occurs *in vitro*. The reaction also occurs in yeast RNA. The optimum conditions for both reactions were pH 5 and high bisulfite concentration. However, by extrapolation, Shapiro (1977) calculates that a concentration of 3×10^{-7} M bisulfite, under physiological conditions, is enough to double the background rate of mutagenesis in mammalian DNA. Also, while it is true that mammalian DNA is double stranded, during the transcriptional process there are regions of single strandedness in the DNA which may be susceptible to this sort of attack. Other subtle conformational and chemical differences in mammalian DNA may exist *in vivo* which may make certain regions of the chromatin more open to such an agent.

A second reaction mechanism of bisulfite *in vitro* which may also result in an alteration of gene function has been described by Shapiro (1977). This reaction is a bisulfite-catalyzed linkage of nucleic acid residues with amino acids and proteins, called transamination. It occurs maximally at pH 7 and high bisulfite concentration. Again, this is thought to be a cytosine-specific reaction, and only single-stranded DNA reacts. A cross-link of DNA with protein or an amino acid would have severe effects on the functioning of the gene.

A third reaction mechanism of bisulfite with nucleic acid is the addition reaction of bisulfite to uracil. The equilibrium constant for addition to uracil is 1×10^3 liters/mol at 25°C, whereas the equilibrium constant for addition to thymidine is 0.3 liter/mol. Thus, this reaction may be specific for RNA and result in a direct inhibition of protein synthesis, interruption of mitosis, or interference with genetic repair. Braverman and Shapiro have shown that 1×10^{-2} M bisulfite blocks the induction of β-galactosidase in *Escherichia coli*, suggesting direct inactivation of protein synthesis. In another study by the same investigators, polyuridylic acid (poly U) was modified by bisulfite at varying concentrations. The ability of poly U to serve as a messenger for polyphenylalanine in a cell-free protein-synthesizing system was then tested. At 10^{-3} M bisulfite, the poly U became 2 percent saturated, yet it retained only 53 percent of its messenger function.

The final reaction mechanism is the free-radical-induced cleavage of internucleotide bonds in DNA by bisulfite. This reaction requires oxygen and Mn^{+2} and is rapid at room temperature at pH 7. It is also inhibited by hydroquinone, a scavenger of free radicals. The degradation occurs during the autoxidation of bisulfite catalyzed by Mn^{+2} ions. Only low concentrations of the active species, $\cdot SO_3^-$ and $\cdot HO_2^-$, are generated during the autoxidation. At higher concentrations the ionic reactions of bisulfite are favored and the autoxidation of bisulfite is quenched. Since this free-radical chain reaction occurs at such low concentrations, it may be of true toxicological importance.

The mutagenic specificity of bisulfite has been demonstrated in bacteria, viruses, yeast, and cultured mammalian cells. Mutant reversion studies on _E. coli_ suggest that bisulfite causes back mutation only in those mutants which had cytosine-guanine pairing at the site of mutation. The number of reversions was optimal at pH 5.2, which presumably reflects a compromise between bacterial viability and a sufficient quantity of bisulfite in the un-ionized form to cross the bacterial wall and membrane. Also, it should be pointed out that although bisulfite does not generally affect double-stranded DNA _in vitro_, it has mutagenic activity in _E. coli_ DNA, which is double stranded.

The mutagenic properties of bisulfite have also been demonstrated in phage λ and T4 bacteriophage. A 10-fold increase in the rate of mutation was found in phage λ exposed to 3 M bisulfite at an acidic pH. The inactivation and mutation of T4 is due to a complete shift of guanine-cytosine to adenine-thymine, induced by bisulfite. A 2- to 20-fold increase in the mutation rate of the yeast Saccharomyces cerevisiae was observed at 5 x 10^{-3} M bisulfite and pH 3.6. The molecular nature of the mutation has not been reported.

Mammalian oocyte damage due to low-level concentrations of bisulfite has been studied by Jagiello et al. (1975). Mouse oocytes exhibited inhibition of meiosis when exposed to 10^{-4} M bisulfite, and chromosomal clumping was observed in oocytes treated with 10^{-3} M bisulfite. Chromosomes of cow oocytes were damaged and meiosis inhibited by treatment with 2.5 x 10^{-2} M sodium bisulfite. Meiosis of oocytes from the ewe exhibited chromosomal aberrations, but meiosis was not inhibited at 2.5 x 10^{-3} M.

Given the reaction mechanisms discussed above, bisulfite may be mutagenic to mammalian cells in vivo. The prerequisite of these reactions is that enough bisulfite be presented to the cell in a form that will allow it to enter the cytoplasm or nucleus of the cell. This capacity is a function of the state of bisulfite in the blood. That is, its ability to enter the cell will depend on the degree of ionization and the amount of bisulfite bound to plasma proteins.

A.2.2.3.2 Reaction with proteins. The chemical disposition of bisulfite in blood is an important aspect of its ability to react with other body tissues and fluids, as previously discussed. Bisulfite reacts reversibly with plasma proteins by sulfitolysis of disulfide bonds:

$$\text{R-S-S-R'} + \text{HSO}_3^- \rightleftarrows \text{R-S-S-O}_3^- + \text{R'SH}$$

The equilibrium constant for this reaction, with cystine in an isolated system at 37°C and pH 7.75, is 8.9 x 10^{-2}. The reaction is essentially complete at low sulfite concentrations. After a 45-min incubation of 54 nmol of sulfite in 480 µl of rabbit plasma under physiological conditions, nearly 100 percent of the bisulfite existed as S-sulfonates, provided the capacity of sulfite-reactive groups was not exceeded.

On the basis of the above findings, Kaplan et al. (1974) have calculated that under chronic sulfur dioxide exposure, approximately 0.16 percent of total plasma proteins would be sulfonated. The alterations in protein structure induced by sulfonation, although reversible, may seriously impair enzyme function. On the other hand, Gunnison and Palmes (1973) and Shapiro (1977) concluded that 0.16 percent is a relatively low level of protein alteration and that sulfitolysis induced by environmental concentrations of sulfur dioxide is probably not metabolically significant. S-sulfonation may serve merely as a vehicle of distribution and storage for bisulfite in the body.

Regardless of whether S-sulfonation of cysteine residues has a significant direct effect on overall enzyme function, these residues could react with other biomolecules. For instance, cytidine and thymine pyrophosphate, compounds with which bisulfite is known to react specifically, failed to react with cysteine S-sulfonate under a variety of experimental conditions. Since bisulfite exists almost entirely as S-sulfonates in the plasma *in vivo*, the negative results of these experiments bring into question the threat of mutagenesis from sulfur dioxide inhalation. Therefore, a number of investigators suggest that the plasma proteins may serve to sequester bisulfite and that the reaction is actually a protective phenomenon. A more recent model on the distribution and turnover of plasma sulfite/bisulfite was reported by Gunnison and Palmes (1976). In their study, sulfite clearance was calculated using plasma decay data obtained from one intravenous injection of sulfite.

By measuring the urinary sulfite following the intravenous administration and using the model conversion rates, they showed that sulfite clearance occurs predominantly by metabolism to sulfate. Their finding indicates that sulfite clearance depends primarily on the efficiency of sulfite oxidase. This enzymatic oxidation may occur within the intermembranous spaces of the mitochondria. They conclude that the sulfate produced in vivo from injected sulfite is not instantaneously accessible to the plasma because the appearance of plasma sulfate lags behind the disappearance of sulfite after equilibrium.

A.2.2.3.3 Reaction with lipids. The interaction of bisulfite with lipids, though not well understood, could greatly alter the structure and function of cellular membranes and upset hormonal activities in vivo.

Bisulfite initiates lipid oxidation in a model system consisting of a Tween 80-corn oil emulsion in water. The oxidative effects of 0.5 to 10 mM bisulfite were assayed for thiobarbituric acid-reactive material. A dose-response relationship for the reaction was observed, and the reaction was quenched by BHT (2,6,di-t-butyl-4-hydroxymethyl phenol), an antioxidant, and 10^{-3} M Mn^{+2}. The inhibition of the autoxidation of lipids by BHT and Mn^{+2} suggests a free-radical mechanism.

Pulmonary surfactant is a complex lipid composed primarily of dipalmitoyl phosphatidylcholine (DPL), which is required for normal respiration. SO_2 promotes the hydrolysis of DPL in vitro by an unknown mechanism. Twenty to 40 percent hydrolysis has been observed. Should such a degradation occur in vivo, pulmonary edema and atelectasis might occur.

Finally, Parkes and Eling (1975) found no effects of 500 ppm sulfur dioxide on the synthesis and degradation of prostaglandins in vitro.

A.2.2.3.4 Reaction with pyridine and flavins. The ionic reaction of bisulfite with NAD^+ and flavins has been described by a number of authors. Bisulfite adds reversibly to the 4 positions of NAD^+ with $K = 36\ M^{-1}$ at pH 7. This adduct was found to be greatly stabilized in the presence of protein since a stoichiometric reaction of bisulfite occurs in low concentrations with protein-bound NAD^+. A similar enhancement of the binding constant of bisulfite with flavins associated with proteins has been observed.

Two reaction mechanisms of bisulfite with NADH have been described. The first is a slow ionic reaction in which bisulfite acts as a general acid catalyst to hydrate the 5,6 double bond. The second reaction is a free-radical oxidation of NADH to the pyridinium salt. It is inhibited by superoxide dismutase, hydroquinone, and EDTA, which suggests a requirement for trace metals.

A.2.2.4 Protective mechanisms--It is evident from the previous discussion that bisulfite is very reactive with a wide range of biomolecules in vitro. The manifestation of the effects of such reactions in vivo depends on the molecule's ability to reach appropriate sites of reactivity and the ability of the body to counter the threat through chemical transformation of bisulfite or sequestration of the molecule by serum protein until it can be removed by urinal excretion. The latter mechanism has already been described.

One method that the body may have to protect itself from the effects of sulfur dioxide intoxication is its ability to oxidize bisulfite to the less toxic sulfate ion. A microsomal liver enzyme, sulfite oxidase, has been purified and characterized. This enzyme, which also occurs in the lung, contains two types of prosthetic groups which perform separate electron transfer functions. The overall reaction is:

$$SO_3^{=} + A + H_2O \rightarrow SO_4^{=} + AH_2$$

This oxidative mechanism may help protect the body from endogenous sulfites produced in normal metabolism, as well as consumed or inhaled sulfites. However, lung levels appear to be low, and saturation of the enzyme with inhaled SO_2 could occur. Also, since this enzyme is subject to end-product inhibition--i.e., inhibition by the sulfate formed--it could also be inhibited by sulfate inhaled along with SO_2 (Rajagopalan and Johnson 1977).

A.2.3 SO_3-H_2SO_4-X_2SO_4

In the atmosphere, it is possible for sulfur dioxide to undergo a catalytic oxidation to sulfur trioxide in the presence of sunlight or metallic oxides. This species can be readily hydrated to sulfuric acid, bisulfite, and sulfate, either in the atmosphere or within the bronchial tree. It has been shown that the sulfate particles so formed are of a size that will deposit predominantly on the gas-exchange regions of the lungs. Inhalation of these particles may result in a variety of pulmonary pathophysiological symptoms. Few studies at the cellular and biochemical level have been performed to elucidate the nature of the effects of sulfate inhalation. (Under conditions of high humidity, atmospheric SO_2, either as aerosol droplets of aqueous solutions or adsorbed on the surfaces of particulates, also undergoes reduction by light in the visible region or by metals such as manganese to produce the

SO_2 free radical which is extremely persistent due to resonance and may be inhaled as such [Gause et al. 1977].)

A.2.3.1 Respiratory tract transport and absorption--The isolated, ventilated, and perfused rat lung has been used to study the kinetics of sulfate transport across the pulmonary and capillary membranes. The $t_{1/2}$ (time) of removal of sodium sulfate was approximately 8.4 min at doses of 0.5 μmol/lung, and this removal occurred by a process of simple absorption. The absorption kinetics were greatly influenced by the cationic species associated with the sulfate anion. The absorption was enhanced with ammonium ion, Co^{+2}, Ni^{+2}, Zn^{+2}, Cd^{+2}, Fe^{+2}, and Hg^{+2}, but not with Mn^{+2}. Furthermore, the process was unidirectional in that absorption from the vasculature to the airways did not occur.

A.2.3.2 Biochemical effects of X_2SO_4--Sulfate salts have also been demonstrated to cause histamine release from lung fragments *in vitro*, and this has been postulated as a mechanism of bronchoconstriction observed *in vivo*. A synergistic action of sulfate and its cationic species has been postulated, since sodium sulfate does not cause a marked release of histamine, whereas ammonium sulfate does. Ammonium chloride, however, results in only a 50 percent release of histamine as compared with ammonium sulfate. These results suggest that entry into cells is important for the release of histamine and that a change in the cationic species may permit an alteration in membrane permeability to sulfate and thus alter the effects of pollutant on the lungs.

The rate of removal of sulfate from the airways also depends upon the pH of the solution containing the sulfate salt. Values removed from the physiological pH of 7.4 on either the acidic or basic side promote sulfate removal. There is a similarity in ranking of different sulfate salts in their rate of removal from the airways in the presence of metal ions and in their bronchoconstrictive potency. Sulfuric acid aerosols are also more potent than sodium sulfate in production of bronchoconstriction, and, similarly, acid solutions of sulfate salts are more readily removed than are neutral solutions. The flux of sulfate ions through the pulmonary mast cells may be the determining factor in the potency of different sulfate salts observed *in vivo*.

Chronic exposure conditions are difficult to produce *in vitro*. An interesting hypothesis dealing with the basic mechanism of inflammation has proposed a central role for superoxide radical anion, $\cdot O_2^-$. Superoxide may promote chronic inflammation through its action on lysosomes and on intracellular organelles containing degradative enzymes, and by the production of chemotactic factors, attractive agents for inflammatory cells. The release of histamine described above is likely to produce only a transient bronchoconstriction. Sulfate ions have recently been shown to stimulate the production of superoxide through a mechanism involving intact peritoneal macrophages. Pulmonary macrophages containing superoxide-generating systems are similar to those described for peritoneal macrophages, and thus sulfate salt inhalation could stimulate pulmonary inflammation independent of the acidic nature of the aerosol. This free-radical form of SO_2 is quite long-lived in both biochemical and atmospheric chemistry model systems. According to Gause et al. (1977), this SO_2 free radical could be important in the radiomimetic effects of SO_2 and in protein thiol-disulfide and lipid-peroxidation reactions. Chronic release of histamine is also likely to stimulate the attraction of other inflammatory cells from the blood and to sustain a local inflammatory response. Pulmonary inflammation is thought to underlie many of the chronic lung diseases seen in man, and these may be linked through such mechanisms.

A.2.4 H_2S and COS

Hydrogen sulfide (H_2S) is a noxious gas with an odor characteristic of rotten eggs. It is heavier than air and is soluble in both water and non-polar solvents. At a physiological pH of 7.4, roughly two-thirds of the hydrogen sulfide exists as HS^-, while only trace amounts exist as divalent sulfur ($S^=$), as seen by its acid dissociation constants:

$$H_2S \rightleftarrows HS^- + H^+ \qquad pK_a = 7$$
$$HS^- \rightleftarrows S^= + H^+ \qquad pK_a = 12$$

Historically, most studies reported on the effects of hydrogen sulfide on hemoglobin. These studies focused on in vitro blood exposure to H_2S, causing a reversible reaction product, sulfhemoglobin--a combination of sulfur with heme. Unfortunately, many of these early studies are unreliable, and later work is confusing. This problem arises from the lack of knowledge of the chemistry of sulfhemoglobin, which has never been observed following in vivo exposure.

Sulfhemoglobin is formed from the reaction of the prosthetic groups of ferric myoglobin binding with sulfur after a sequential reaction with peroxide and hydrogen sulfide. It occurs also from the binding of the sulfide to the heme iron.

Several biochemical lesions have been reported following in vitro exposure of H_2S, particularly on respiratory chain electron transfer. Submitochondrial studies indicate that sulfide is more potent than cyanide in inhibiting cytochrome aa_3, the terminal enzyme in the respiratory chain. This mechanism could be responsible for the toxic effects caused by acute high-concentration exposures. Sulfide binds to cytochrome aa_3 regardless of the cytochrome redox state. The undissociated sulfide-cytochrome species is a more active inhibitor than the anionic species.

Generally, the toxicologic mechanisms of hydrogen sulfide seem to parallel those of cyanide; both substances inhibit cytochrome aa_3. In addition, sulfide can also inhibit red blood cell glutathione peroxidase, the enzyme involved in protecting the cell from oxidative damage. A number of other enzymes, such as horseradish peroxidase, potato "polyphenol oxidase," uricase, amine oxidase, and catalase, can also be inhibited by sulfide.

Carbonyl sulfide (carbon oxysulfide [COS]) was discovered by Than through the reaction of sulfuric acid on potassium thiocyanate. Because COS hydrolyzes rapidly in water, forming carbon dioxide and hydrogen sulfide, it occurs at only relatively low concentrations from fossil fuel burning. As seen in the summary table of Chapter 2, COS is emitted in only trace amounts from mobile sources, primarily during failure modes.

Carbonyl sulfide is discussed briefly in the in vitro literature as a metabolic product of carbon disulfide. In a study by Dalvi et al. (1975), carbon disulfide (CS_2) was incubated with rat liver microsomes in the presence of NADPH, producing COS. COS was then metabolized by the mixed-function oxidase enzyme system to carbon dioxide. In a reaction analogous to the metabolism of CS_2 to COS, COS then released its sulfur atom, which bound to the microsomes. This binding mechanism inhibited benzphetamine metabolism and decreased the concentration of cytochrome P-450.

Although COS is shown *in vitro* only to inhibit drug metabolism, specifically benzphetamine, Dalvi et al. also note that COS might combine with trace metals such as copper and zinc in animal systems.

COS also affects the nervous system in various animal species; however, there are no *in vitro* data explaining this mechanism.

A.2.5 Summary

Studies of the properties of the hydrated forms of sulfur oxides point to a number of potential toxic actions. Sulfur dioxide on hydration forms the bisulfite ion, which is highly reactive, especially with the cell's genetic material. There is little doubt that bisulfite produces significant degradation of both DNA and RNA and that these modifications can result in mutations. Bisulfite also reacts readily with serum proteins and probably with cellular proteins. The net effect of sulfitolysis on catalytic proteins such as enzymes is not clear from the experiments performed to date. Inhibition, stimulation, and no effect have been described from this reaction. Sulfitolysis of serum proteins may be a protective mechanism reducing the amount of mutagenic bisulfite ion that is free in the blood to diffuse into cells and to react with cellular genetic mechanisms. Detoxification enzymes exist in the liver to remove bisulfite from the blood. Variations in the activity of these detoxifying enzymes may affect the ability of an individual to resist chronic SO_2 exposure. Whether SO_2 inhalation at present atmospheric concentrations is likely to lead to greater mutations in man cannot be determined from the presently available data.

Sulfate aerosols produce bronchoconstriction in animals and man apparently due to the release of histamine by sulfate salts, probably through an ionic mechanism within the cell. The chemical nature of sulfate aerosols in the atmosphere is not known, but the potency of different sulfate salts to promote bronchoconstriction *in vivo* or the release of histamine *ex vivo* and *in vitro* suggests that different chemical compositions of the natural aerosols are likely to produce large differences in their health effects. Some of the more potent sulfate salts are those likely to be found in the smaller (<3 μm) particles, and the acidity of the aerosol is also highly important. Studies using model systems such as the isolated, ventilated, and perfused lung have proven the utility of measurements *ex vivo*; many of the transient effects could not be measured *in vivo*. Lastly, the chronic effects of sulfate inhalation are likely to involve cell-mediated inflammation.

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A.3 IN VIVO

A.3.1 Introduction

Chronic low-level exposure to sulfur-bearing compounds in the ambient air may have significant effects on human health. However, relatively little research has been done on animals to explore these effects for compounds other than SO_2 or H_2SO_4. Most _in vivo_ work has utilized short-term exposure schedules, and these studies have been limited, for the most part, to pollutant effects on cardiopulmonary function. Thus, vast areas remain to be explored--long-term exposure regimens, the impact of other sulfur-bearing compounds present in the atmosphere, and the various other physiological parameters that may be affected. The following discussion deals with animal exposure studies that have been conducted to date. For convenience, the presentation is organized by the types of compounds studied, including sulfur dioxide, sulfuric acid, sulfates and sulfites, and pollutant combinations. The textual discussion of these various compounds is supplemented by summary tables, which appear at the end of this section.

A.3.2 Sulfur Dioxide

Over a short period of exposure, only extremely large concentrations of sulfur dioxide are lethal. In fact, studies have shown that brief exposure to a concentration of the gas exceeding the ambient air level by a thousand times may not prove fatal. Extended exposure to relatively high concentrations may, however, result in death. For example, a continuous-exposure study showed that a concentration of 130 ppm SO_2 took 154 hr to kill half the guinea pigs exposed. Mice were more resistant, and it required 847 hr of continuous exposure at a 150-ppm concentration to kill half their population. At exposures of about 1000 ppm, survival time was greatly reduced and species sensitivity was reversed. At this concentration, half the mice died within 4 hr, and half the guinea pigs died within 20 hr (Hazelton Laboratories Report 1969b).

Leong et al. (1961) exposed animals to extremely high concentrations of SO_2 (up to 5000 ppm). Some of the subjects were pretreated with histamine to simulate respiratory disability. Others had been adrenalectomized to simulate the condition of humans with low resistance to nonspecific stress. Both treatments shortened survival time as compared with that of a control group. The lung pathology observed postmortem was primarily dependent on exposure duration. In animals that had not been pretreated, pulmonary edema and areas of consolidation were evident. In both the histamine-pretreated and adrenalectomized animals, bronchial obstruction was seen. In guinea pigs that succumbed rapidly, there was occlusion of the bronchioles and venous congestion with little or no fluid in the alveoli. Examination of animals that survived for 2 to 4 hr revealed fluid in the alveoli, distended bronchioles, and partial desquamation of the mucosal membranes.

SO_2 is an upper respiratory tract irritant that is absorbed readily by the mucous membranes of the upper airway. At relatively high concentrations, most of the SO_2 that is inspired is absorbed in the upper respiratory airways and thus prevented from directly reaching the deep lungs. Only at low concentrations is this absorption effect reduced. Strandberg (1964) demonstrated this phenomenon in rabbits, as indicated below.

SO_2 concentration mg/m^3	SO_2 concentration ppm	Absorption, %	Range, %
26	100	95	90-98
52	20	95	75-95
13	5.0	80	50-90
1.3	0.5	50	10-60
0.26	0.1	5	1-10

Source: Strandberg (1964).

However, SO_2 is delivered in a dose-related manner by two routes, inhalation and the circulatory system. For example, Frank and Speizer (1965) noted that radiolabeled SO_2 absorbed in the upper airway entered the blood and thereby reached the lung.

Shortly after inhalation, SO_2 decreases respiratory rate and narrows the bronchial tubes, resulting in an increase in airway resistance. This acute effect is reversible. Corn (1972) and Frank (1965) noted that SO_2 concentrations greater than 1 ppm generally increased pulmonary flow resistance in various animal species. The effects were dose related and diminished shortly after cessation of exposure. Amdur (1973) found that 1 hr of exposure to as little as 0.5 ppm SO_2 increased pulmonary flow resistance in guinea pigs. The increase peaked at about 10 percent and abated after the exposure period.

- Among the various species that have been tested, guinea pigs show the longest persistance of SO_2 effects on air flow. Amdur (1961) observed the following effects after a 1-hr exposure:

SO_2 concentration, mg/m^3	Duration of increased flow resistance following exposure, hr
260	1
780	2
800	>2

Source: Amdur (1961).

Alarie (1973) administered 17 ppm SO_2 to mice and noted a decrease in respiratory rate. After repeated exposures lasting 3-10 min each, the effect disappeared. Wakisaka (1975) examined SO_2-induced changes in respiratory rate in two groups of mice: one group had been exposed to SO_2 previously, and the other had no prior exposure. The animals previously exposed showed less of a decline in respiratory rate.

Other effects may result from acute high doses of SO_2. For example, Knauss et al. (1976) exposed rats continuously to 600-700 ppm SO_2 and found an increase in tracheal mucus after 9 hr. However, Spiegelman et al. (1968) did not observe any effect on bronchial clearance in donkeys exposed for 30 min to SO_2 in the dose range of 25-713 ppm.

Lee et al. (1966) exposed guinea pigs to 7-20 ppm SO_2 for 2-1/2 hr and found that hemoglobin increased in linear proportion to the dose. This relationship was not found at higher exposure levels (21-320 ppm).

Long-term exposures to SO_2 show varying results, depending on concentration, duration, and animal species. Generally, relatively high concentrations are required to produce a chronic response in animals, such as alteration of pulmonary structures and ventilatory function. As in the case of acute effects, chronic effects appear in animals only at concentrations far above ambient air levels.

Hirsch et al. (1975) exposed beagles to 1 ppm SO_2 twice daily for 1-1/2 hr over a 12-month period. No significant changes in respiratory mechanics or pulmonary function were observed. A similar finding was obtained by

Alarie (1970, 1972), who exposed guinea pigs continuously to 1-5 ppm SO_2 for 1 year and monkeys to 0.5-1.3 ppm for 78 weeks. Lewis et al. (1969), however, found changes in the respiratory mechanics of dogs after 225 days of continuous exposure to 5 ppm SO_2. These changes included increased pulmonary flow resistance and decreased lung compliance (a measure of lung distensibility). Asmundsson et al. (1973) found that exposing animals to SO_2 concentrations of 40 or 100 ppm for 6 weeks continuously did not produce bronchial damage, but that such damage did occur when a 200-ppm concentration was used.

Significant deterioration in pulmonary function was also observed in monkeys (Alarie 1972) after an accidental high dose of SO_2. Following a 30-week continuous exposure to 5 ppm SO_2, a dose of 200-1000 ppm was inadvertently administered to the test animals for approximately 90 min. The subjects exhibited dilation of the bronchioles and chronic dilatation of the bronchi. These effects did not occur when Alarie (1975) repeated the original experiment, involving continuous exposure to 5 ppm for 78 weeks.

Weiss and Weiss (1976) noted alterations in pulmonary function in mice following continuous exposure for 6-9 days to 40 ppm SO_2. In addition to an increase in static lung compliance and a decrease in lung surface tension, there was a 21 percent weight loss.

Reid (1963) exposed rats to 300-400 ppm SO_2 for 3 months and observed a hypersecretion of mucus, an effect which simulated one of the conditions of chronic bronchitis in humans. In addition to increased numbers of mucous cells in the main bronchi, mucous cells also were noted in the peripheral airways, where they are normally absent. Dalhamn et al. (1956) exposed rats continuously to 10 ppm SO_2 for 6 weeks and observed a thickening of the mucous layer. The cilia could not clear away the mucus because of its thickness, and the clearance of foreign bodies from the respiratory system was consequently decreased. Fraser et al. (1968) did not find a change in ciliary activity after exposing rats to 300 ppm SO_2 for 12 hr daily over 4 months.

Chakrin et al. (1974) observed changes in the goblet cells of bronchi and bronchioles in beagles exposed to 500-600 ppm SO_2 for 2 hr twice a week for 4-5 weeks. These changes produced a pus-like exudate in the bronchial tree.

A.3.3 Sulfuric Acid

Sulfuric acid mist rapidly disperses into dilute suspensions and reacts with ions in the atmosphere, such as ammonia, to form atmospheric sulfate salts. The H_2SO_4 contained in an aerosol is particulate matter, and its toxicity therefore depends upon its deposition and retention in the respiratory tract. Sulfuric acid particles in the submicron size range are deposited at a high rate by the mechanism of Brownian movement, although the rate depends also on relative humidity. As compared with other sulfate particles, H_2SO_4 particles are more numerous and have greater surface area per unit volume. Green and Lane (1964) note that foreign nuclei, such as salts, in hygroscopic vapors may affect the size, and hence the toxicity, of the aerosol. These factors contribute to the toxic potential of the compound. In studies of both short-term exposure (Amdur 1970) and long-term exposure (Alarie 1973, 1975), sulfuric acid has been found to be the most irritating of the particulate sulfur species. Amdur (1978) observed that equimolar amounts of H_2SO_4 more than tripled the increase in pulmonary flow resistance caused by SO_2.

Treon (1950) observed that H_2SO_4 toxicity varied among four animal species. These animals, in order of increasing sensitivity, were: rabbits < rats < mice < guinea pigs. This variation may have arisen from differences in respiratory function and structure. However, it has also been suggested that guinea pigs are particularly susceptible because of their high histamine content. Cockrell et al. (1976) noted that exposure to 100 mg/m^3 H_2SO_4 for 2 hr was fatal to guinea pigs, while the same concentration did not kill rats even after 6 months of exposure.

Animal health and age may also influence the toxic response to H_2SO_4. Amdur et al. (1952) noted that among guinea pigs 1-2 months old, half could not survive an 8-hr exposure to 18 mg/m^3 H_2SO_4, while among animals 18 months old, a concentration of 50 mg/m^3 was required to achieve a 50 percent mortality.

Sackner et al. (1977) exposed animals to 1 mg/m^3 H_2SO_4 for 10 min and found no change in respiratory flow. However, unlike SO_2, sulfuric acid may cause damage not reflected in respiratory frequency changes. For example, Amdur et al. (1975) found that H_2SO_4 altered lung resistance and compliance at concentrations which did not decrease breathing rate.

In a short-term study, Amdur (1958) found an increase in flow resistance and a decrease in lung compliance in guinea pigs after 1 hr of exposure to various H_2SO_4 aerosols differing in concentrations (0.2-40.0 mg/m^3) and particle size (7, 2.5, 0.8, and 0.3 μm). These changes occurred as the concentration increased and the particle size decreased.

A recent study by Amdur (1978) examined the irritant effects of H_2SO_4, several inorganic sulfates, and pollutant combinations with SO_2 in guinea pigs. One-hour exposures of 1 mg/m^3 H_2SO_4 and less at particle sizes of 1 and 0.3 μm caused significant increases in resistance. The smaller particles produced a greater response at a given concentration than the 1-μm particles. Amdur noted that the response to 0.1 mg/m^3 with 1-μm particles was slight and rapidly reversible, while the same concentration with 0.3-μm particles was greater in magnitude and not rapidly reversible (see Figure A-1).

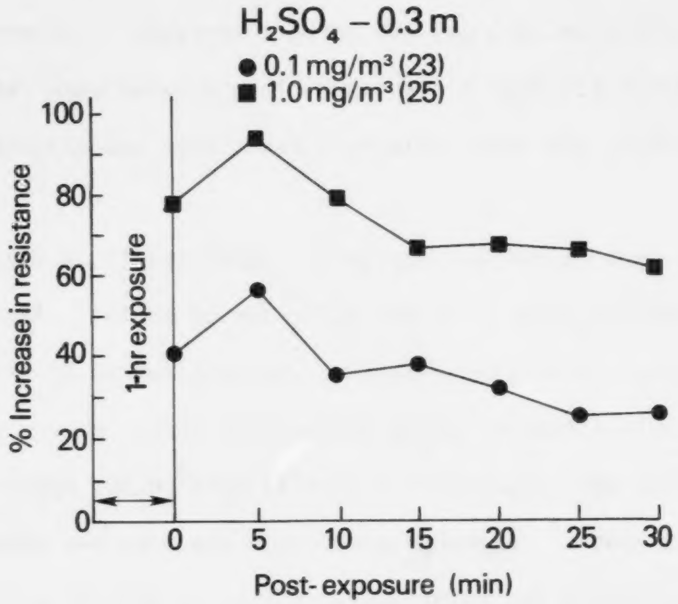

Figure A-1. Post-exposure changes in pulmonary flow resistance produced by 0.3-μm sulfuric acid. Numbers in parentheses are numbers of animals exposed.

Source: Amdur (1978).

In chronic studies by Amdur et al. (1975), flow resistance increased with exposure to H_2SO_4 within a concentration range of 0.07-0.86 mg/m^3 for 1-μm particles. The irritant potency also increased for H_2SO_4 particles ranging from 0.1-2.5 μm, the size which can be deposited in the gaseous exchange regions of the lung. Following exposure to 0.4-0.6 mg/m3 concentrations made up of particles in this size range, lung resistance and compliance values were altered.

Long-term continuous exposures to low concentrations of $H2^{SO}4$ do not appear to affect pulmonary function to the same degree as short-term exposures to high concentrations. Lewis et al. (1975) continuously exposed dogs to 800 $\mu g/m^3$ H_2SO_4 (particle size: 0.5 µm) and noted a decrease in pulmonary function after 225 days. After 621 days of exposure, lung volumes were reduced, heart weights were decreased, and total pulmonary resistance was elevated.

Alarie (1975) exposed guinea pigs to 0.1 mg/m^3 (particle size: 2.78 µm) and 0.08 mg/m^3 (particle size: 0.84 µm) H_2SO_4 for 12 months. Although effects in pulmonary mechanics, diffusing capacity, and ventilation distribution were found, comparison with a control group showed that these changes were not significant. Monkeys were exposed to 0.38-4.79 mg/m^3 H_2SO_4 (particle size: 0.54-3.6 µm) for 18 months. Although breathing frequency was elevated, lung compliance and inspiratory and respiratory resistance did not differ significantly from that of control group animals. However, over 78 weeks of exposure to H_2SO_4, monkeys exhibited deleterious effects on pulmonary structures (Alarie 1973). The critical concentrations were 4.79 mg/m^3 with 0.73-µm particles and 2.73 mg/m^3 with 3.60-µm particles.

Generally, the differences noted between short-term exposures to high doses and chronic exposures to low doses might be explained by the presence of ammonia in the upper airways. Larson (1977) suggested that ammonia may mitigate the effects of inhaled acid sulfate, particularly at low concentration levels.

A.3.4 Sulfates and Sulfites

Suspended sulfate levels have been associated with cardiopulmonary symptoms in the elderly and asthmatics. However, there are not sufficient data to quantitatively link ambient air levels with health effects. It is still not known whether the metal salts themselves or the acid precursors of these salts are the actual irritants.

Inhalation studies with animals have shown that sulfates are more irritating to the respiratory system than SO_2 alone or in the presence of an insoluble salt. The various sulfate salts that have been studied have different irritant potencies. The following compounds may be ranked in order of irritant potency on a scale of 1 to 100: H_2SO_4, 100; zinc ammonium sulfate, 33; ferric sulfate, 26; zinc sulfate, 19; ammonium sulfate, 10; ammonium bisulfate, 3; copper sulfate, 2; ferrous sulfate, 0.7; sodium sulfate, 0.7; manganous sulfate, -0.9 (not significant). These rankings were based on studies with guinea pigs (Amdur 1978).

Amdur (1978) examined the comparative irritant potency of ammonium sulfate, ammonium bisulfate, copper sulfate, and sodium sulfate. The concentrations and sizes of the salts were $(NH_4)_2SO_4$, 0.5-9.5 mg/m^3, 0.13-0.81 µm (MMD); NH_4HSO_4, 0.9-10.9 mg/m^3, 0.13-0.77 µm (MMD); $CuSO_4$, 0.4-2.4 mg/m^3, 0.11-0.33 µm (MMD); and Na_2SO_4, 0.9 mg/m^3, 0.11 µm (MMD). Ammonium sulfate caused a statistically significant decrease in compliance at all concentrations and particle sizes. Except for a minimal response to the 0.2-µm particles, the response per microgram of sulfate increased as particle size decreased. Ammonium bisulfate showed significant changes in resistance and compliance, but was less irritating than ammonium sulfate. Copper sulfate at 0.4 mg/m^3 (0.1 μm) produced a slight but significant change in compliance but not in resistance. At twice this concentration, changes in resistance and compliance were observed for 0.1- and 0.3-μm particles. The degree of response was less with the larger particles. Sodium sulfate did not significantly change resistance or compliance. A further discussion of these sulfate salts appears in the pollutant combination section.

Nadel et al. (1965) exposed anesthetized cats to 40-50 mg/m^3 zinc ammonium sulfate (particle size: 0.1-1.5 μm) for 3 months. Pathological examination showed a narrowing of the bronchioles, which correlated with increased pulmonary resistance, decreased compliance, and increased end-expiratory transpulmonary pressure.

Bell and Hackney (1976) exposed squirrel monkeys to 2.5 mg/m^3 ammonium sulfate for 1 hr and found a significant increase in flow resistance. In another short-term study, Sackner et al. (1976) exposed dogs to 4.6 mg/m^3 ammonium sulfate for 75 min and found no pulmonary changes within 30 min after the exposure period. They did find, however, that a smaller exposure (0.94 mg/m^3) exerted effects within 1-2 hr after the termination of exposure: while no change in flow resistance or functional residual capacity was noted, there was a 20 percent decrease in lung compliance.

Bell and Hackney (1977) presented priority criteria for restudying sulfate salts. Charlson et al. (1977) noted that the most abundant sulfate aerosols are those formed by the neutralization of H_2SO_4 and NH_3, such as ammonium sulfate, ammonium bisulfate, and the mixed salt $(NH_4)_3H(SO_4)_2$. It was also noted that the bisulfate ion (HSO_4^-) may be intrinsically more irritating than the sulfate ion ($SO_4^=$) in an aqueous solution. Particle size may be an important factor in the irritancy of sulfate salts. For example, in guinea pigs, Amdur and Corn (1963) found an increase in flow resistance with decreasing mean particle diameter at 1-mg/m^3 concentrations of zinc ammonium sulfate (Figure A-2).

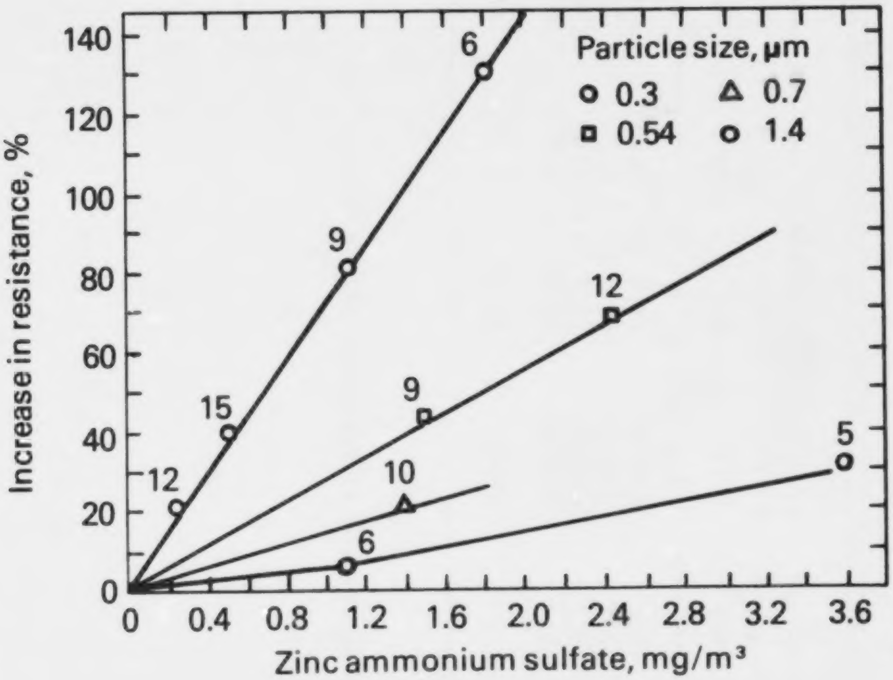

Figure A-2. Dose-response curve of zinc ammonium sulfate aerosol for different particle sizes. Numerals beside each point indicate the number of animals.

Source: Amdur and Corn (1963).

The toxicology of inhaled metal sulfites has not been studied extensively. However, it has been found that sodium metabisulfite is a sensory irritant when inhaled by mice. Alarie et al. (1973) noted that at 1 ppm, the metabisulfite affected the respiratory rate to a greater extent than did SO_2. At this concentration, sodium sulfite was inactive.

Table A-1. MORTALITY AND SURVIVAL RATE OF MICE EXPOSED FOR 3 HR TO SULFATES AND CHALLENGED WITH STREPTOCOCCUS AEROSOL

SO_4,	$ZnSO_4$			$Zn(NH_4)(SO_4)_2$			$(NH_4)_2SO_4$		
	Mortality			Mortality			Mortality		
mg/m^3	D/T[b]	%	MST,[a] day	D/T	%	MST,[a] day	D/T	%	MST,[a] day
0	373/1689	22.1	12.1	165/756	21.8	12.3	233/588	39.6	10.1
≦1.1	125/599	20.9	12.2	29/120	24.2	11.9	22/48	45.8	10.1
1.2-2.0	369/813	45.4[c]	9.1[c]	112/445	25.2	12.0	76/191	39.8	10.7
2.1-3.0	186/278	66.9[c]	6.2[c]	67/192	34.9[c]	10.9[c]	52/96	54.2[c]	9.2
3.1-4.0	-	-	-	-	-	-	47/144	32.6	11.2
≧4.1	-	-	-	45/48	93.8[c]	4.0[c]	46/110	41.8	10.3

[a] MST = Mean survival time.

[b] D/T = Dead/total.

[c] Significant change from infected mice exposed to filtered air ($P \leqq 0.05$).

When McJilton et al. (1973) exposed guinea pigs to an aerosol containing SO_2 and sodium chloride, they noted an increase in pulmonary flow resistance only at high relative humidity (80 percent). This effect could have been associated with the presence of a bisulfite ion, which was noted in the chemical analysis of the aerosol.

In studying the combined effects of SO_2 and various sulfate salts, Amdur (1975) found that a combination including copper sulfate exhibited the greatest degree of synergism. It was noted, however, that since divalent copper readily forms sulfite complexes, the observed effects may have resulted from a copper sulfite aerosol.

In an inhalation study, Ehrlich et al. (1978) investigated the susceptibility to respiratory infection of mice exposed for 3 hr to filtered air, zinc sulfate, ammonium sulfate, or zinc ammonium sulfate, followed by a respiratory challenge with viable bacteria (Streptococcus pyogenes). Approximately 90 percent of the particles were less than 3 μm in diameter. Table A-1 shows the mortality and survival rates of the test animals. No deaths were noted among mice exposed to sulfate aerosols only. The death rates for control mice challenged with S. pyogenes averaged 22.1 percent. Using regression analysis, Ehrlich et al. calculated a concentration relationship between the metal sulfate salts and enhanced mortality caused by the infection. A zinc sulfate concentration of 1.45 mg/m^3 was required to induce 20 percent excess mortality, while a 2.40-mg/m^3 concentration of zinc ammonium sulfate was required to produce the same effect. Ammonium sulfate did not affect susceptibility to infection.

A.3.5 Pollutant Combinations

The combination of air pollutants, such as exists in the ambient atmosphere, appears to exert greater effects on human health than do individual pollutants. Animal studies have demonstrated the operation of a synergistic principle in the effects of these combinations.

Amdur (1961) and Amdur and Underhill (1968) studied the effects of SO_2 in combination with various "inert" aerosols (i.e., not injurious at the experimental levels tested). For example, they exposed animals to various concentrations of the sulfur compound along with sodium chloride (NaCl) aerosol at a 10-mg/m^3 concentration. They observed that when NaCl aerosol was present, not only was the response to SO_2 greater, but also the return to control values was delayed.

Amdur (1968) found that in guinea pigs a combination of SO_2 and various salts (manganese chloride, ferrous sulfate, and sodium orthovanadate) produced effects on mechanical function which exceeded those produced by SO_2 alone and which were totally absent when only the salts were present. The author hypothesized that soluble metal salts can catalyze the conversion of SO_2 to sulfuric acid, which causes a synergistic (or exaggerated) response. Thus, a possible explanation for this action is that a gas-aerosol reaction occurred in the high relative humidity of the upper respiratory tract. To generalize from this example, such a reaction would depend on the solubility of SO_2 in the aerosol of a given salt.

Amdur (1978) combined SO_2 exposure with exposure to the four sulfate salts noted previously. In all the exposures to guinea pigs, SO_2 at 0.3 ppm and sulfate particle sizes of 0.1 μm, there was a statistically significant change in resistance and compliance. Ammonium sulfate, ammonium bisulfate, and sodium sulfate did not potentiate the response to SO_2. However, copper sulfate, a relatively mild irritant compared with other sulfate salts, gave more than an additive effect with SO_2. Figure A-3 shows the combination and individual exposures.

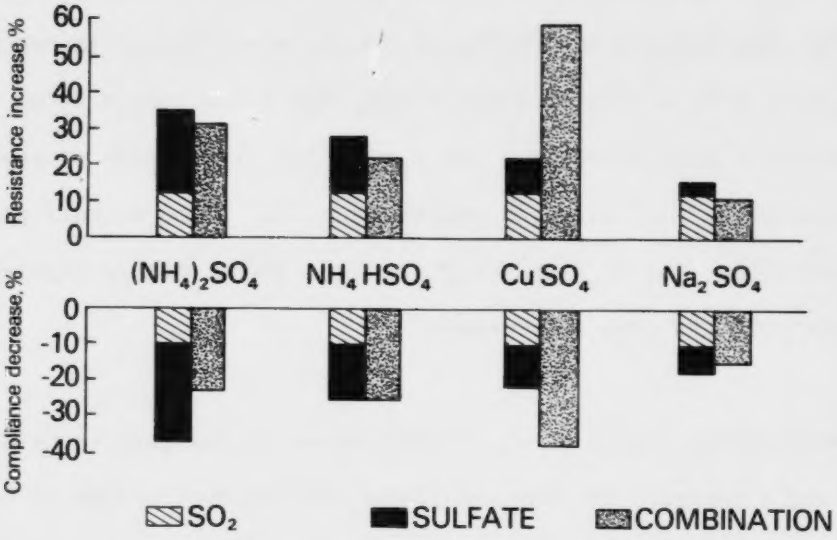

Figure A-3. Comparison of responses to combinations of SO_2 and sulfates with the sum of the responses to each given alone.

Source: Amdur (1978).

Hyde et al. (1976), Gillespie et al. (1976), and Lewis et al. (1974) studied the combined effects of SO_2 and auto exhaust on dogs. Lewis et al. found that a 2-year exposure to raw exhaust and SO_2 at an approximate concentration of 1.1 mg/m^3 produced increased total lung capacity. Irradiated auto exhaust alone and with SO_2 increased respiratory resistance.

Gillespie et al. compared pulmonary function measurements obtained at the conclusion of the exposure period with measurements made 2 years later. All the dogs, except for those breathing filtered air, showed an increased total lung capacity. Flow resistance was elevated in animals exposed to irradiated and nonirradiated exhaust in combination with SO_2 and H_2SO_4. It was concluded that alterations in pulmonary function occurred even after termination of the exposure period. Gillespie et al. and Hyde et al. further observed that dogs exposed to irradiated exhaust exhibited an increase in flow resistance, with a loss of dynamic lung compliance. Hyde et al. examined the lungs of these dogs 2-1/2 to 3 years after the termination of exposure. They observed minimal increases in enzyme production among animals exposed to sulfur oxides alone, but no significant increase among animals exposed to sulfur oxides combined with auto exhaust.

In another study, Lewis et al. (1969) showed an increase in pulmonary resistance and a decrease in lung compliance and residual volume in beagles. These effects arose from a continuous exposure for 225 days of H_2SO_4 mist in combination with SO_2 at concentrations of 0.8 and 1.3 mg/m^3, respectively.

Gardner et al. (1977) examined the effect of H_2SO_4 (2-hr exposure to a 900-µg/m^3 concentration with particles of 0.23-µm volume mean diameter) and/or ozone (3-hr exposure to a 196-µg/m^3 concentration) on susceptibility to pulmonary infectious disease in mice. After exposure, the animals were challenged with an aerosol of viable bacteria (Streptococcus pyogenes) and were returned to clean air for a 15-day holding period. Exposure to either the acid or ozone alone had no effect on mortality, nor did a sequential exposure first to the acid and then to ozone. However, as shown in Table A-2, when the animals received sequential exposure first to ozone and then to the acid, significant mortality effects occurred.

Table A-2. PERCENT MORTALITY (TREATED-AIR CONTROL) FOR VARIOUS EXPOSURE REGIMENS

	Exposure regimen					
Summary data	Air + H_2SO_4	H_2SO_4 + Air	Air + O_3	O_3 + Air	H_2SO_4 + O_3	O_3 + H_2SO_4
Number of experiments	9	10	9	10	11	10
Average % mortality	2.2	6.0	6.1	7.0	5.0	10.8
Standard error	5.0	5.7	5.9	4.8	4.4	4.2

Source: Gardner et al. (1977).

Amdur (1978) exposed guinea pigs for 2 hr to 0.2, 0.4, or 0.8 ppm O_3 singly or in combination with equal concentrations of SO_2. The two higher ozone concentrations caused a decrease in compliance. The SO_2 exposures did not significantly change resistance. The O_3/SO_2 combination produced responses similar to that of O_3 alone. Thus, no interactive effects were observed from this pollutant combination. When the animals were exposed to 1 or 10 ppm SO_2 and motor oil mist (10 mg/m^3), the irritant effects of SO_2 (resistance increase) were antagonized. Mineral oil (medicinal-grade naphthalene), however, did not counter the SO_2 effects. But when the detergent or dispersant component used in the motor oil was added to the mineral oil, a protection against SO_2 was observed. Lastly, the addition of alpha-tocopherol to the mineral oil also protected against SO_2. The study did not attempt to determine whether this was a local effect in the lung or an effect that could be produced if the vitamin was administered orally or by injection.

Laskin et al. (1970) exposed rats and hamsters to SO_2 alone and combined with benzo(a)pyrene (BP) to determine carcinogenesis from inhaling two combined urban air pollutants. In isolation exposure chambers, rats and hamsters were exposed to SO_2 for 6 hr/day, 5 days/week. An equal number of control animals received prefiltered air only. Animals from each group were transferred to an internal inhalation chamber for the BP/SO_2 exposure of 1 hr/day, 5 days/week (Table A-3).

Table A-3. INHALATION EXPOSURES OF RATS AND HAMSTERS TO SULFUR DIOXIDE AND/OR BENZO(a)PYRENE ATMOSPHERES[a]

Exposure type	Exposure pattern, 5 days/week		Exposure period, 794 calendar days Exposure (days)	
	Irritant, 6 hr/day	Carcinogen-irritant, 1 hr/day	I	CI
A (fresh air)				
A + CI		10 mg/m^3 BP + 3.5 ppm SO_2	534	494
I	10 ppm SO_2			
I + CI	10 ppm SO_2	10 mg/m^3 BP + 3.5 ppm SO_2	534	494

[a] Isolated living quarters contained prefiltered, conditioned fresh air during nonexperimental periods.

No significant pathology was found in hamsters, but squamous cell carcinoma was noted in rats (Table A-4). Of the 21 rats exposed to SO_2 (I) and carcinogen-irritant (CI), 5 had squamous cell carcinoma of the lung appearing after 547 to 794 calendar days of exposure. The earliest cancer showed local extension and metastasis to the kidney. Two additional animals had advanced squamous metaplasia 485 and 715 days.

Table A-4. INHALATION EXPOSURES TO SULFUR DIOXIDE AND/OR BENZO(a)PYRENE ATMOSPHERES

Exposure type	Number of animals	Number with advanced squamous metaplasia[a]	Number with squamous cell carcinoma[a]
A	3	0/3	0/3
A + CI	21	1/21	2/21
I	3	0/3	0/3
I + CI	21	2/21	5/21[b]

[a] Expressed as a ratio of tumors found to animals observed.
[b] Secondary squamous cell carcinoma in kidney.

Table A-5 shows some correlations between results of *in vitro* and *in vivo* studies of sulfur-bearing compounds.

Table A-5. COMPARISONS BETWEEN IN VITRO AND IN VIVO STUDIES OF SULFUR-BEARING COMPOUNDS

Chemical	Effects: In vitro	Effects: In vivo
$Zn(NH_4)_2(SO_4)_2$	Changes in alveolar macrophages	Increased susceptibility to disease in mice
$SO_4=$	Histamine release from lung fragments	Bronchoconstriction
SO_2/HSO_3-	Mutagenesis in cultured mammalian cells	With BP, pulmonary carcinogenesis in rats
$MnSO_4$, Na_2SO_4	No substantial histamine release	No bronchoconstriction
$MnSO_4$	No enhanced absorption with manganese cations	No effects of pulmonary mechanics
$(NH_4)_2SO_4$	Marked increase in histamine release	Bronchoconstriction
SO_2 + NaCl	Deposited to more distal parts of a simulated lung	Increased changes in pulmonary mechanics
SO_2 + $CuSO_4$	Divalent copper readily forms sulfite complexes	Caused more than an additive effect on pulmonary function
SO_2/HSO_3-	Increased ATPase activity in rat alveolar macrophages	Increased ATPase and lysozyme activity in baboon alveolar macrophages
SO_2/HSO_3-	SO_2 absorbed in a simulated tracheobronchial system	In most animals, >90% SO_2 is absorbed in upper airways
SO_2/HSO_3-, $SO_4=$	Greater amounts of histamine release in guinea pigs	Higher sensitivity to SO_2 + $SO_4=$ aerosols in guinea pigs

Table A-6. SUMMARY TABLE: DOSE EFFECTS OF SULFUR-BEARING COMPOUNDS ON VARIOUS ANIMAL SPECIES

Chemical	Concentration, mg/m^3	Time	Species	Effect
H_2SO_4	0.1-1.0	30 min	Guinea pig	Increased pulmonary resistance
H_2SO_4	1	2 hr	Hamsters Rabbits	Decreased ciliary activity; hematological changes, but no immune changes
H_2SO_4	2.4 and 4.8	2 hr	Monkeys	Pulmonary function changes and alteration of lung structure
$ZnSO_4$ or $Zn(NH_4)_2(SO_4)$	1.3 or 2.1	3 hr	Mice	Increased susceptibility to infection (dose-response effects)
$(NH_4)_2SO_4$	1-≥4.1	3 hr	Mice	No increased susceptibility to infection
SO_2 with and without carbon particles	5.2	102 days 192 days	Mice	Changed immune system
H_2SO_4 on carbon particles	1	3 hr/day, 5 days/wk, 20 wk	Mice	Altered immune system and lung structure
$O_3 + H_2SO_4$	0.2 + 1.0	3 hr + 2 hr	Mice	Increased susceptibility to infection
$Zn(NH_4)_2(SO_4)_2$	1.1	1 hr	Guinea pig	81% increase in pulmonary resistance
$ZnSO_4$	0.9	1 hr	Guinea pig	40% increase in pulmonary resistance
NH_4SO_4	1.0	1 hr	Guinea pig	22% increase in pulmonary resistance
$FeSO_4$	1.0	1 hr	Guinea pig	77% increase in pulmonary resistance
$Fe_2(SO_4)_3$	1.0	1 hr	Guinea pig	2% increase in pulmonary resistance
$MnSO_4$	4.0	1 hr	Guinea pig	No increase in pulmonary resistance

Table A-6 (Continued). SUMMARY TABLE: DOSE EFFECTS OF SULFUR-BEARING COMPOUNDS ON VARIOUS ANIMAL SPECIES

Chemical	Concentration, mg/m^3	Time	Species	Effect
SO_2 + NaCl	0.41-26.0	1 hr	Guinea pig	Increased pulmonary resistance, tidal volume, and frequency of breathing; decreased minute ventilation
SO_2	2.6	2 hr	Monkeys	No observed effects
SO_2	2.6-26.0	20-40 min	Dogs	Increased pulmonary resistance; decreased lung distensibility
SO_2 + O_3	0.5-2.1 + 0.4-1.6	2 hr	Guinea pig	No interactive effects from pollutant combination

Table A-7. SUMMARY TABLE: EXPOSURE TO SULFUR DIOXIDE

Author	Species	Substance	Concentration		Duration	Response
			mg/m^3	ppm		
Frank and Speizer (1965)	Dog	SO_2 (exposure via: nasal route, tracheal cannula, surgically isolated segments of trachea)	18.2-1398	7-230	15-min periods	Nasal: Increased pulmonary flow resistance proportional to exposure concentration at SO_2 levels ranging from 18.2-158.6 mg/m^3 (7-61 ppm). Progressively greater response at 65 mg/m^3 (25 ppm) and above. Isolated tracheal segments: Increased flow resistance of the lungs at high concentrations of SO_2. Bronchoconstriction and increased flow resistance were more pronounced when lung itself was exposed to SO_2 than when upper airways alone were exposed.
Lee and Danner (1966)	Guinea pig	SO_2	18.2-806	7-310	2-1/2 hr	Increase in hemoglobin concentration; this was linear with increase in SO_2 concentration between 18.2 and 52 mg/m^3 (7 and 20 ppm) only.

Table A-7 (Continued). SUMMARY TABLE: EXPOSURE TO SULFUR DIOXIDE

Author	Species	Substance	Concentration		Duration	Response
			mg/m^3	ppm		
Frank et al. (1967)	Dog	$^{35}SO_2$	49.2	22	Not stated	95% of $^{35}SO_2$ was absorbed by the mucosa; $^{35}SO_2$ was present in the lungs via pulmonary capillaries; ^{35}S was present in the blood and urine within 5 min of exposure.
Peacock and Spence (1967)	Mouse	SO_2	1300	500	5 min/day, 5 days/wk for 300 days	SO_2 increased the incidence of primary lung tumors by 50% over the controls; SO_2 exposure hastened the onset of pulmonary neoplasias.
Spiegelman et al. (1968)	Miniature donkey	SO_2	67.6-1882.8	26-713	30 min	Below 780 mg/m^3: No impairment in bronchial clearance. Above 780 mg/m^3: Slowing or transient arrest of clearance due to increased mucus secretion.
Hazelton Laboratories Report (1969b)	Guinea pig	SO_2	338 ~2600	130 ~1000	154 hr 20 hr	LD_{50} LD_{50}
	Rat	SO_2	390 ~2600	150 ~1000	847 hr 4 hr	LD_{50} LD_{50}

Table A-7 (Continued). SUMMARY TABLE: EXPOSURE TO SULFUR DIOXIDE

Author	Species	Substance	Concentration mg/m^3	Concentration ppm	Duration	Response
Frank et al. (1969)	Dog	$^{35}SO_2$	2.6-260	1-100	5-30 min	99% of SO_2 inhaled through the nose is absorbed, while 95% of the SO_2 inhaled through the mouth is absorbed and kept from entering the lungs.
Alarie et al. (1970)	Guinea pig	SO_2	0.26 2.60 13.00	0.1 1.0 5.0	22 hr/day, 7 days/wk continuously for 1 year	No detrimental change in pulmonary function.
Barry and Mawdesley-Thomas (1970)	Rat	SO_2	720	300	6 hr/day for 10 days	Increase in acid phosphatase activity in free alveolar cells, indicating an increase in infiltration of mucus in the peribronchial region.
Laskin et al. (1970)	Rat	SO_2	26 132 273 1474.3	10 51 105 567	6 hr/day, 5 days/wk for: 113 days 112 days 22 days 12 days	1474.3, 273 mg/m^3 (567, 105 ppm): Exposure induced mortality. 87 and 40% occurred within 12 and 22 days, respectively. Dead animals showed congestion, edema, pneumonia. Gross pulmonary damage and clinical symptoms were absent at 26 mg/m^3 (10 ppm) and most marked at 1474.3 mg/m^3 (567 ppm). Tracheitis, bronchitis, and
	Rat and hamster	SO_2	26.2	10	6 hr/day, 5 days/wk for 794 days	

Table A-7 (Continued). SUMMARY TABLE: EXPOSURE TO SULFUR DIOXIDE

Author	Species	Substance	Concentration mg/m^3	Concentration ppm	Duration	Response
	Group I	SO_2, then SO_2 + B(a)P	26.2 9.17 10	10 3.53 3.85	6 hr/day, 5 days/wk, then 1 hr/day, 5 days/wk for 794 days	ulceration of trachea and bronchi were observed at 273 and 1474.3 mg/m^3 (105 and 567 ppm). Hamster mortality was completed by 98 weeks of exposure, and no significant pathology was found. Group I: 5/21 rats developed squamous cell carcinomas of the lung; 2 additional animals showed advanced squamous metaplasia. The earliest cancer showed local extension and renal metastasis. Group II: 2/21 rats developed squamous cell carcinomas of the lung; both animals showed renal metastasis. A third rat showed advanced squamous metaplasia.
	Group II	Filtered air, then SO_2 + B(a)P	- 9.17 10	- 3.53 -	1 hr/day, 5 days/wk for 794 days	
Yokoyama et al. (1971)	Dog	$^{35}SO_2$	57.2 130	22 50	30-60 min	Greatest concentration of ^{35}S found in the dialyzable fraction was associated with alphaglobulins; urinary ^{35}S was in the form of inorganic sulfate; ^{35}S remained in blood stream for 3 hr post-exposure.

Table A-7 (Continued). SUMMARY TABLE: EXPOSURE TO SULFUR DIOXIDE

Author	Species	Substance	Concentration mg/m^3	Concentration ppm	Duration	Response
Alarie et al. (1972)	Cynomolgus monkey	SO_2	0.36 1.67 3.33 12.2 + 520-2600	0.14 0.64 1.28 4.69 + 200-1000	24 hr/day for 78 wk 24 hr/day for 30 wk + 90 min	Exposure to 0.36-3.33 mg/m^3 had no deleterious effects on pulmonary function. After exposure to 12.2 mg/m^3 for 30 wk, 90-min exposure to 520-2600 mg/m^3 caused decreases in pulmonary function and pathological changes.
Giddens and Fairchild (1972)	Mouse (male)	SO_2	26	10	Varying periods up to 72 hr	Severe injury (lesions) in nasomaxillary turbinates of respiratory tract.
Hirsch (1975)	Beagle	SO_2	2.6	1.0	1-1/2 hr twice a day for 12 months	No significant changes in respiratory mechanics or pulmonary function were observed.
Roth and Tansey (1972)	Rat	SO_2	780	300	2 hr	Gastric inhibition.
Alarie et al. (1973)	Mouse	SO_2	0-744.8 312	0-298 120	10 min 10 min, 12, 7, or 3 min. Recovery, 10 min more.	SO_2: Immediate decrease in respiratory rate, then progressive return to pre-exposure rate. With successive exposures, desensitization to SO_2.

Table A-7 (Continued). SUMMARY TABLE: EXPOSURE TO SULFUR DIOXIDE

Author	Species	Substance	Concentration mg/m^3	Concentration ppm	Duration	Response
		SO_2	260-325	100-125	90 sec/exposure for 10 exposures	CBMN: Rapid respiratory depression, slows to equilibrium, then rapid recovery at end of exposure. No desensitization.
		O-chlorobenzilidene malononitrile (CBMN)	75 2.4	- -	10 min 10 min	CBMN + SO_2: No additive effect; response equal to CBMN alone. Prior exposure to either substance does not change response to SO_2 or CBMN.
		CBMN + SO_2	75 260	- 100	Not stated	Maximum sensory irritation by SO_2 of upper respiratory tract occurs at 1-2 min of exposure. Desensitization of SO_2 is peculiar to this substance.
Callanan et al. (1975)	Rabbit	SO_2	520	200	2 min	Coughing immediately upon exposure. Bronchoconstriction and increase in flow resistance followed within 1 min post-exposure. Hering-Breuer reflex (responsible for automatic inflation and deflation of the lungs) was lost or reduced in most subjects. The rabbits were vagotomized and re-exposed. Following exposure, pulmonary stretch receptors were inactivated and the Hering-Breuer reflex was lost in most subjects.

Table A-7 (Continued). SUMMARY TABLE: EXPOSURE TO SULFUR DIOXIDE

Author	Species	Substance	Concentration		Duration	Response
			mg/m^3	ppm		
Antweiler and Brockhaus (1976)	Guinea pig (female)	SO_2 NO_2 $SO_2 + NO_2$	10 10 10 each	3.85 - -	Continuously, 6 days/wk for 6 months	Neither SO_2, NO_2, nor a combination of the two caused any significant changes in respiratory frequency, flow rate, minute volume, or body weight.
Knauss et al. (1976)	Rat (female)	SO_2	1560-1820	600-700	3 hr/day, 6 days a week. Examined after 0, 9, 18, 30 hr of cumulative exposure	Tracheal mucus retention and increases in amount of solid material (cellular and mucous) positively correlated with exposure time. Bronchial solid increase due to inflammatory cells; goblet cells in seconday bronchi declined at onset but increased after 30-hr exposure ($P < 0.01$).
Weiss and Weiss (1976)	Mouse	SO_2	104	40	Continuous exposure for 6-9 days	Exposed mice lost 21% of body weight; static lung compliance increased 37% ($P < 0.01$). Alveolar stability, lung weight, and lung wet/dry weight ratio decreased 2 to 6%; trapped gas increased but not statistically significantly. Also, surface tension decreased.

Table A-7 (Continued). SUMMARY TABLE: EXPOSURE TO SULFUR DIOXIDE

Author	Species	Substance	Concentration mg/m³	Concentration ppm	Duration	Response
Amdur (1961)	Guinea pig	SO_2	>260-2080	>100-800	1 hr	Reported in terms of duration of pulmonary flow resistance following exposure: Doses less than 260 mg/m³ (>100 ppm), ~1 hr; 780 mg/m³ (300 ppm), >2 hr; 2080 mg/m³ (800 ppm), <2 hr.
Amdur (1966)	Guinea pig	SO_2	0.416	0.16	1 hr	10% increase in pulmonary flow resistance following exposure.
Reid (1963)	Rat	SO_2	780-1040	300-400	Daily exposure for 3 months	Increased number of mucus cells were produced in the main bronchi and peripheral airways.
Dalhamn et al. (1956)	Rat	SO_2	26	10	Continuous exposure for 6 wk	Thickening of mucus layer, causing a decrease in clearance of foreign bodies from respiratory system. Mucus was too thick to be moved efficiently by cilia.
Goldring et al. (1967)	Syrian hamster	SO_2	1690	650	3 hr/day, 75 days	No increase in respiratory infection in 75-day-exposed animals.
Fairchild et al. (1972)	Mouse (already infected with influenza)	SO_2	0-52.0	0-20	7 days continuous	Effects observed at 18.2-26 mg/m³ (7-10 ppm). Exposed mice developed more pneumonia than controls. Associated with lung lesions due to SO_2 exposure.

Table A-7 (Continued). SUMMARY TABLE: EXPOSURE TO SULFUR DIOXIDE

Author	Species	Substance	Concentration mg/m^3	Concentration ppm	Duration	Response
Hatch et al. (1974)	NAMRU mouse	SO_2, then *Klebsiella pneumoniae* challenge	(1) <0.2	<0.08	4 hr	(1) Increased resistance to to infection.
			(2) >0.2	>0.08	4 hr	(2) Decreased resistance to infection.
			(3) <0.2	<0.08	72 hr	(3) Decrease resistance to infection.
Amdur (1978)	Guinea pig	SO_2	0.832	0.32	~1 hr	Significant change in resistance and compliance.

Table A-8. SUMMARY TABLE: EXPOSURE TO SULFURIC ACID

Author	Species	Substance	Concentration, mg/m^3	Duration	Response
Treon et al. (1950)	Rabbit Rat Mouse Guinea pig	H_2SO_4 mist (95% of the particles were <2 μm in diameter)	87-1600	N/A	Exposure to H_2SO_4 particles revealed species differences in sensitivity to toxic effects. Sensitivity increased as follows: rabbits < rats < mice < guinea pigs.
Amdur et al. (1952)	Guinea pig	H_2SO_4 mist (particles had a mass median diameter of 1 μm)	18	8 hr	The indicated exposure represented the LC_{50} for animals 1-2 months old.
			50	8 hr	The indicated exposure represented the LC_{50} for animals 18 months old.
			8	8-72 hr	Mortality rates did not vary within the exposure range indicated, suggesting that concentration is more important than duration in H_2SO_4 lethality. However, pulmonary pathology was greatly increased with the longer exposure.
Thomas et al. (1958)	Guinea pig	H_2SO_4 mist of three particle sizes: 0.6, 0.9, and 9.0 μm	≧ 4.0	18-140 days	The greatest degree of pulmonary pathology occurred on exposure to particles of 0.9-μm diameter. In young animals, this mist produced

Table A-8 (Continued). SUMMARY TABLE: EXPOSURE TO SULFURIC ACID

Author	Species	Substance	Concentration, mg/m^3	Duration	Response
					slight pulmonary edema and occasional hemorrhage from pulmonary capillaries. Mucus production was decreased in the bronchi, and there was some evidence of increased desquamation of epithelial cells in smaller bronchi, though the bronchial epithelium throughout the lungs appeared intact. Exposure to the 9.0-μm particles resulted in upper respiratory tract pathology, including slight laryngeal and tracheal edema.
Bushtueva (1962)	Guinea pig	H_2SO_4 aerosol	0.5-2.0	5-14 days	Exposure to 2 mg/m^3 for 5 days resulted in pulmonary edema and thickening of alveolar walls. Exposure to 1 mg/m^3 for 1 to 2 wk resulted in slight tracheobronchial catarrh and infiltration of lymphoid cells around the blood vessels and bronchi. Exposure to 0.5 μg/m^3 resulted in "slight lung irritation."

Table A-8 (Continued). SUMMARY TABLE: EXPOSURE TO SULFURIC ACID

Author	Species	Substance	Concentration, mg/m^3	Duration	Response	
Amdur (1975)	Guinea pig	H_2SO_4 aerosol of 5 particle sizes:		1 hr	Pulmonary flow resistance	
					% Increase	% Increase / mg $SO_4^=/m^3$
		0.1 µm	0.07		32	432
		0.3 µm	0.1		41	410
		0.7 µm	0.12		43	358
		1.0 µm	0.07		14	205
		2.5 µm	0.22		18	84
Amdur (1958)	Guinea pig	H_2SO_4 particles (particle size: 0.3-7 µm)	0.2-40	1 hr	Increased pulmonary flow resistance and decreased lung compliance as concentration rose and particle size decreased. Smaller particles produced a faster onset of response.	
Fairchild et al. (1975)	Mice	(1) H_2SO_4 aerosol (particle size: 3.2 µm)	0.45 and 15.0	90 min/day for 4 days	(1) Only concentrated H_2SO_4 (15 mg/m^3, 3.2 µm) reduced nasal clearance of nonviable streptococci.	
		^{33}P-labeled Streptococcus aerosol (particle size: 0.6 µm)	1.5	30 min (after the last H_2SO_4 exposure)		

Table A-8 (Continued). SUMMARY TABLE: EXPOSURE TO SULFURIC ACID

Author	Species	Substance	Concentration, mg/m^3	Duration	Response
		(2) H_2SO_4 aerosol (particle size: 3.2 µm)	1.5 and 15	One 4-hr exposure	(2) Only concentrated H_2SO_4 (15 mg/m^3, 3.2 µm) reduced nasal and lung clearance.
		^{33}P-labeled *Streptococcus* aerosol (particle size: 0.6 µm)	1.5	N/A	No exposure regimen reduced rate of clearance of viable streptococci.
Amdur (1978)	Guinea pig	H_2SO_4 (particle size: 0.3-1 µm)	0.1-1.0	~1 hr	Resistance values at all concentrations increased. Greatest response was produced by 0.3-µm particles.

Table A-9. SUMMARY TABLE: EXPOSURE TO SULFATES AND SULFITES

Author	Species	Substance	Concentration mg/m^3	Concentration ppm	Duration	Response
Alarie et al. (1973)	Mouse	Na_2SO_3 (sodium sulfite)	36.4 93.6 293.8 795.6	14 36 113 306	10 min	No effect from sodium sulfite. Rapid decrease in respiratory rate with exposure to $Na_2S_2O_5$. Response is similar to that from SO_2 exposure. At low concentrations, response is slightly higher to $Na_2S_2O_5$ than to SO_2.
		$Na_2S_2O_5$ (sodium metabisulfite)	65 195 254.8 494 780	25 75 98 190 300	10 min	
Gunnison and Palmes (1973)	Rabbit	SO_3=(intravenous)	0.9 μmol/kg of body wt		-	Rapid formation of plasma S-sulfonate (peak at 20-30 min post-injection). Half of this was cleared about 1 hr after injection.
		SO_3=(ingested)	8.9 or 25 μmol/ml		-	
		SO_2 (inhaled)	26 mg/m^3	10	3-10 days	
						S-sulfonate increased during exposure until reaching equilibrium at about 3-5 days. Clearance of S-sulfonate proceeded for a number of days after exposure was concluded. Clearance of S-sulfonate was much slower after SO_2 inhalation than from sulfite ingestion and much faster for short-term intravenous exposure to sulfite.

Table A-9 (Continued). SUMMARY TABLE: EXPOSURE TO SULFATES AND SULFITES

Author	Species	Substance	Concentration, mg/m^3	Duration	Response
Nadel et al. (1965)	Cat	$ZnSO_4(NH_4)_2$-$SO_4 \cdot 6H_2O$	40-50	3 min	Pulmonary flow resistance increased in response to particles of 0.1-1.5 μm, as indicated by bronchioli narrowing to <400 μm.
Amdur and Underhill (1968)	Guinea pig	$Fe_2(SO_4)_3$	1.0	1 hr	77% increase in pulmonary flow resistance.
		$FeSO_4$	1.0	1 hr	No effect on pulmonary flow.
		$MnSO_4$	1.0	1 hr	No effect on pulmonary flow.
Amdur (1978)	Guinea pig	$(NH_4)_2SO_4$ (particle size: 0.13-0.81 μm)	0.363-6.926	1 hr	Significant decrease in compliance at all concentrations and particle sizes.
		NH_4HSO_4 (particle size: 0.13-0.77 μm)	0.775-9.157		Increase in resistance and decrease in compliance. Less irritating than $(NH_4)_2SO_4$.
		$CuSO_4$ (particle size: 0.13-0.77 μm)	0.257-1.448		0.4 mg/m^3 (0.1-μm diameter) produced a slight decrease in compliance. 2 mg/m^3 (0.1-μm diameter) increased resistance and decreased compliance. Particles 0.3 μm in diameter produced less change than the smaller particles. No statistically significant changes in resistance or compliance.
		Na_2SO_4 (particle size: 0.1 μm)	0.608		No statistically significant changes in resistance or compliance.

Table A-10. SUMMARY TABLE: EXPOSURE TO SULFUR DIOXIDE + PARTICULATES, AEROSOLS, AND H_2SO_4

Author	Species	Substance	Concentration, mg/m^3	Duration	Response
Fraser et al. (1968)	Rat	SO_2	2.62-7.86	12 hr/day, 7 days/wk for 4 months	No alteration in ciliary activity.
		SO_2 +	2.62-7.86 + 1		No alteration in ciliary activity.
		Graphite dust (particle size: 1.5 μm)	1		
Lewis et al. (1969)	Beagle (female)	H_2SO_4 mist	0.9		Decreased diffusion capacity.
		SO_2	13.4		Decreased lung compliance and increase in flow resistance.
		SO_2 +	13.4	21 hr/day, 225 days for both	Smaller residual volume than controls.
		H_2SO_4 mist	0.9		
Amdur and Underhill (1968)	Guinea pig	SO_2	52 (20 ppm)	1 hr	
		Soluble salts			
		NaCl	10		Insoluble salts elicited no increased response; soluble salts of Mg, Fe++, V potentiated response to SO_2, increasing flow resistance more rapidly than NaCl or KCl.
		KCl	10		
		$MgCl_2$	1.0		
		Na_3VO_4	0.7		
		Fe_2O_3	11.7	2 hr	
			21.0		
		Insoluble particles			
		Spectrographic carbon	8.0		
		Activated carbon	8.7		
		MnO_2	9.7		
		Open hearthdust	8.0		

Table A-10 (Continued). SUMMARY TABLE: EXPOSURE TO SULFUR DIOXIDE + PARTICULATES, AEROSOLS, AND H_2SO_4

Author	Species	Substance	Concentration mg/m^3	Concentration ppm	Duration	Response
Battigelli et al. (1969)	Rat	SO_2	2.62	1	12 hr/day, 7 day/wk for 4 months	No difference between experimental animals and controls exposed to fresh air and graphite dust.
		+				
		Graphite dust	1			
Rylander (1969)	Guinea pig	Coal dust	5.9		6 hr/day for 20 days	Coal dust was effective in reducing clearance; the effect was increased by SO_2.
		SO_2	27.0	10.4		
		Coal dust	5.9			
		+				
		SO_2	13.0	5		
Amdur and Underhill (1970)	Guinea pig	SO_2	3.93-6.81	1.51-2.62	1 hr	No significant effects observed.
		+				
		FeO dust (particle size: 0.76 µm)	1.0-24			
		SO_2	0.42-2.1	0.16-0.81		
		+				
		Open hearth dust (particle size: 0.037 µm)	0.12-0.72			
Rylander et al. (1970)	Guinea pig	MnO_2	5.9		6 hr/day for 20 days	Neither SO_2 nor MnO_2 hindered clearance individually; in combination, bacterial clearance was reduced.
		SO_2	15.6	6.0		
		MnO_2	5.9			
		+	+			
		SO_2	13.0	5.0		

Table A-10 (Continued). SUMMARY TABLE: EXPOSURE TO SULFUR DIOXIDE + PARTICULATES, AEROSOLS, AND H_2SO_4

Author	Species	Substance	Concentration mg/m^3	Concentration ppm	Duration	Response
Corn et al. (1972)	Cat (male)	SO_2	52	20	Not stated	2 of 20 animals showed increase in pulmonary flow resistance after exposure.
		SO_2 +	52	20		
		NaCl aerosol (particle size: 25 µm)	10			
Islam et al. (1972)	Dog	ACh aerosol	N/A			
		SO_2	2.6 5.2 7.8 26	1 2 3 10	Groups of 1, 2, and 3 hr	Increase in bronchial sensitivity. Maximum sensitivity from 5.2 mg/m^3 > 2.6 mg/m^3 > 13 mg/m^3 > 26 mg/m^3. Peak reaction at 3 hr of exposure.
Zarkower (1972)	Mouse	Carbon (particle size: 1.8-2.2 µm)	0.56		Up to 192 days	Decrease in antibody formation in all cases.
		SO_2	5.2	2		
		Carbon +	0.56			
		SO_2	5.2	2		

Table A-10 (Continued). SUMMARY TABLE: EXPOSURE TO SULFUR DIOXIDE + PARTICULATES, AEROSOLS, AND H_2SO_4

Author	Species	Substance	Concentration		Duration	Response
			mg/m³	ppm		
Alarie et al. (1973)	Cynomolgus monkey	H_2SO_4 mist (particle size: 0.4 μm)	0.38-4.79		78 wk continuous	Deleterious effects on pulmonary structures at concentrations of 2.43 and 4.79 μg/m³ with particles of 3.60 and 0.73 μm.
	Guinea pig	H_2SO_4 mist (particle size: 2.8 or 0.8 μm)	0.10 or 0.08		52 wk continuous	No effects observed.
	Cynomolgus monkey	SO_2 + Fly ash	0.26 2.60 13.0 0.5	0.1 1.0 5.0	78 wk	No effects observed.
	Guinea pig	SO_2 + Fly ash	Same as above		52 wk	No effects observed.
Lewis et al. (1973)	Beagle (female)	SO_2 H_2SO_4 aerosol	13.4 0.9	5.15	21 hr/day, 620 days	H_2SO_4 exposure decreased CO diffusion capacity. No effect on hematological index. Decrease in lung and heart weights; decrease in diffusion capacity; deleterious effect on conduction airways and lung parenchyma. (Did not state any effects from combination exposure). No specific effects of SO_2.
		SO_2 + H_2SO_4	13.4 0.9	 5.15		

Table A-10 (Continued). SUMMARY TABLE: EXPOSURE TO SULFUR DIOXIDE + PARTICULATES, AEROSOLS, AND H_2SO_4

Author	Species	Substance	Concentration, mg/m^3	Duration	Response
McJilton et al. (1973)	Guinea pig	SO_2	2.86 (1 ppm)		**Various exposure modes were tested:** (1) SO_2 + low RH (2) SO_2 + high RH (3) NaCl + low RH (4) NaCl + high RH (5) SO_2 + NaCl + low RH (6) SO_2 + NaCl + high RH Significant increased pulmonary flow resistance found only in NaCl particles; rapid uptake of SO_2 and transfer to the respiratory tract.
		NaCl (particle size: <2 µm)	0.91		
		RH	Low, 40% High, 80%		
Krishman et al. (1974)	Albino rat (male)	"Fuming" H_2SO_4 (particle size: <2 µm)	1827.8	Group 1: 5 hr Group 2: 5 hr/day for 6 days Group 3: 5 hr/day for 6 days followed by 24-hr recovery period Group 4: 5 hr/day for 5 weeks	Group 1: No effects observed. Group 2: Increase in lung surfactant activity (decreased maximum and minimum surface tension). Histopathological changes: Emphysematous changes in cells; peribronchial and perivascular inflammatory cell infiltrations. Group 3: No changes observed following recovery period (suggest reversibility of effects from short-term exposure).
		Control (filtered air)	-		

Table A-10 (Continued). SUMMARY TABLE: EXPOSURE TO SULFUR DIOXIDE + PARTICULATES, AEROSOLS, AND H_2SO_4

Author	Species	Substance	Concentration, mg/m^3	Duration	Response
Krishman et al. (continued)					Group 4: Decrease in lung surfactant activity. Histopathological changes: emphysema, alveolar thickening, inflammatory cell infiltration in perivascular and peribronchial regions; congestion; edema observed in 1 animal. Suggests that following extended exposure, production of lung surfactant is inhibited.
Alarie et al. (1975)	Cynomolgus monkey (19 groups of 9 animals)	Group A Control (filtered air)		78 wk	Increased pulmonary flow resistance from exposure to H_2SO_4 + SO_2 and H_2SO_4 + fly ash (group A).
		SO_2	13.1		
		H_2SO_4 + Fly ash	0.11-0.99 0.53-0.55		Groups A and B: Microscopic cellular changes. (a) H_2SO_4 + fly ash: Changes in the mucosa of the bronchi and respiratory bronchioles.
		SO_2 + H_2SO_4 (particle size: 4.11-5.34 μm - fly ash 0.52-1.07 μm - H_2SO_4)	0.28-13.86 0.10-0.97		(b) SO_2 + H_2SO_4: Changes in bronchial mucosa; appearance of focal goblet cell hypertrophy and hyperplasia; some focal areas of squamous metaplasia in other areas of the bronchial tree.

Table A-10 (Continued). SUMMARY TABLE: EXPOSURE TO SULFUR DIOXIDE + PARTICULATES, AEROSOLS, AND H_2SO_4

Author	Species	Substance	Concentration, mg/m^3	Duration	Response
Alarie et al. (continued)		Group B		78 wk	(c) SO + H_2SO_4 + fly ash: Same alterations as in (b). Fly ash did not alter those effects attributed to SO_2 + H_2SO_4. H_2SO_4 is responsible for effects observed; no synergism could be observed.
		Control (filtered air)	-		
		SO_2 + H_2SO_4	0.28-2.89 0.11-0.93		
		H_2SO_4 + Fly ash	0.09 0.45		
		SO_2 + H_2SO_4 + Fly ash (particle size: 0.50-3.35 μm - H_2SO_4 4.10-5.89 μm - fly ash)	0.28-13.86 0.09-0.88 0.41-0.48		
	Guinea pig	Control (filtered air)	-	52 wk	No detrimental effects observed.
		H_2SO_4	0.90		
		H_2SO_4 + Fly ash (particle size: 0.49-2.23 μm - H_2SO_4 3.50-5.31 μm - fly ash)	0.08 0.45-0.46		

Table A-10 (Continued). SUMMARY TABLE: EXPOSURE TO SULFUR DIOXIDE + PARTICULATES, AEROSOLS, AND H_2SO_4

Author	Species	Substance	Concentration	Duration	Response
Charles and Menzel (1976)	Rat	(1) $^{35}S-Na_2SO_4$	0.01 mM 0.10 mM 1.00 mM	1 - 2-sec intervals	(1) Percentage of ^{35}S-absorbed at a given interval was constant; assume absorption occurred by simple diffusion; half-life for sulfate absorption in the presence of sodium is 34.5 min.
		(2) Na_2SO_4 $(NH_4)_2SO_4$ Na_2SO_4 + NaCl Na_2SO_4 + NH_4Cl	0.01 mM 0.01 mM 0.01 mM + 1.00 mM 0.01 mM + 1.00 mM	1 - 2-sec intervals	(2) Sulfate ion absorption increased by 10.8-3.1% in the presence of 0.02 mM NH_4^+ and decreased by 6.6 ± 3.0% in the presence of 1 mM Cl^-; in the presence of 1 mM of both Cl and NH_4^+ the rate was increased by 10.9 ± 2.3%. Absorption was potentiated by the cationic species.
Bell and Hackney (1976)	Squirrel monkey	$(NH_4)_2SO_4$ aerosol (particle size: 0.58 μm MMD) at relative humidity of 40% and 85%	~2.5 mg/m^3	1-hr and 2-hr periods	Small increase in respiratory resistance after 1-hr exposure at 40% relative humidity. Significant changes ($P < 0.5$) in resistance occurred at both 1- and 2-hr exposures at 85% relative humidity.

Table A-10 (Continued). SUMMARY TABLE: EXPOSURE TO SULFUR DIOXIDE + PARTICULATES, AEROSOLS, AND H_2SO_4

Author	Species	Substance	Concentration	Duration	Response
Cockrell et al. (1976)	Rat	H_2SO_4 mist	5 to 100 mg/m^3	6 hr/day for 48 hr to 6 months	No exposure-related pulmonary lesions found in rats at any concentration or exposure duration.
	Guinea pig				Exposure-related lesions were found in guinea pigs exposed to H_2SO_4. Those exposed to 100 mg/m^3 died several hours after exposure.
Sackner et al. (1976)	Dog	Aerosol $NaNO_3$	740 μg/m^3 4000 μg/m^3	75 min	Measurements of functional residual capacity (FRC), static lung compliance (Cl), and total respiratory resistance (Rrs) were taken 5, 15, 30, 60, 120, and 180 min after exposure.
		$(NH_4)_2SO_4$	940 μg/m^3 4560 μg/m^3		
		$NaNO_3$ solution (i.v. injection)	100 mg		
					$NaNO_3$, 740 and 4000 μg/m^3: No change in any parameter.
					$(NH_4)_2SO_4$, 940 μg/m^3: No change in FRC or Rrs; Cl fell 20% between 60 and 120 min ($P < 0.02$). 4560 μg/m^3: Followed up to 30 min post-exposure.
					100-mg $NaNO_3$ injection: No detectable effect on pulmonary and carotid blood pressures, cardiac output, and arterial blood gases.

Table A-10 (Continued). SUMMARY TABLE: EXPOSURE TO SULFUR DIOXIDE + PARTICULATES, AEROSOLS, AND H_2SO_4

Author	Species	Substance	Concentration, mg/m^3		Duration	Response
Lewis et al. (1974)	Beagle	(1) Filtered air			16 hr/day for 61 months	Pulmonary hyperinflation; increased respiratory resistance in the group exposed to irradiated auto exhaust + sulfur dioxide.
		(2) $CO + CH_4 + NO_2$ + BI (non-irradiated exhaust)	CO	112.1 ± 11.5		
			HC[a]	18.0 ± 2.9		
			NO_2	0.09 ± 0.04		
			NO	1.78 ± 0.52		
		(3) $CO + CH_4 + NO_2 + NO$ + (irradiated auto exhaust)	CO	108.6 ± 22.5		
			HC	15.6 ± 4.00		
			NO_2	1.77 ± 0.68		
			NO	0.23 ± 0.36		
			Ox[b]	0.39 ± 0.18		
		(4) $SO_2 + H_2SO_4$	SO_2	1.10 ± 0.57		
			H_2SO_4	0.09 ± 0.04		
		(5) $CO + CH_4 + NO_2 + NO + SO_2 + H_2SO_4$ (non-irradiated auto exhaust + SO_2 + H_2SO_4)	CO	113.1 ± 15.9		
			HC	17.9 ± 2.8		
			NO_2	0.09 ± 0.06		
			NO	1.86 ± 0.54		
			SO_2	1.27 ± 0.61		
			H_2SO_4	0.09 ± 0.04		

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[a] HC as CH_4.
[b] Ox as O_3.

Table A-10 (Continued). SUMMARY TABLE: EXPOSURE TO SULFUR DIOXIDE + PARTICULATES, AEROSOLS, AND H_2SO_4

Author	Species	Substance	Concentration, mg/m^3	Duration	Response
Lewis et al. (continued)		(6) $CO + CH_4 + NO_2 + NO + SO_2 + H_2SO_4$ (irradiated auto exhaust + SO_2 + H_2SO_4)	CO 109 ± 22.8 HC 15.6 ± 3.90 NO_2 1.68 ± 0.68 NO 0.23 ± 0.36 Ox 0.39 ± 0.16 SO_2 1.27 ± 0.61 H_2SO_4 0.09 ± 0.04		
		(7) NO(L) + NO_2(H)[a]	NO_2 1.21 ± 0.22 NO 0.31 + 0.08		
		(8) NO(H) + NO_2(L)	NO_2 0.27 ± 0.62 NO 2.05 ± 0.26		
Gillespie et al. (1976)	Beagle	(1) Filtered air (2) Raw auto exhaust (3) Irradiated auto exhaust (4) SOx (5) Raw auto exhaust + SO_2	See Lewis et al. (1974)	Animals were exposed to the substances for 61 months and then to atmospheric air for 2 yr.	Two years after the termination of a 61-month exposure period, all but the control group showed an increase in total lung capacity over the TLC recorded immediately after the 61 months. The control group showed no change.

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[a] L = low concentration; H = high concentration.

Table A-10 (Continued). SUMMARY TABLE: EXPOSURE TO SULFUR DIOXIDE + PARTICULATES, AEROSOLS, AND H_2SO_4

Author	Species	Substance	Concentration	Duration	Response
		(6) Irradiated auto exhaust (7) NO_2 (high conc) + NO (low conc) (8) NO (high conc) + NO_2 (low conc)			Only small increases were noted in inspiratory volume, vital capacity, and TLC for groups 7 and 8. Pulmonary resistance increased in groups 2, 5, and 6. Groups 3, 5, and 6 showed decreases in lung compliance.
Hyde et al. (1976)	Beagle (female)	See Lewis et al. (1974)	See Lewis et al. (1974)	All 8 experimental groups were exposed to a single regimen for 61 months and to atmospheric air for 2-1/2 to 3 yr following exposure.	There were no significant differences in the amount of collagen found in the exposed lung tissue. Nitrogen oxide environments and irradiated exhaust + SO_2 + H_2SO_4 also showed increased enzyme concentration; sulfur oxides produced minimal increases; auto exhaust + SOx produced no effects.

Table A-10 (Continued). SUMMARY TABLE: EXPOSURE TO SULFUR DIOXIDE + PARTICULATES, AEROSOLS, AND H_2SO_4

Author	Species	Substance	Concentration		Duration	Response
			mg/m^3	ppm		
McJilton et al. (1976)	Guinea pig	SO_2 +	2.86			Changes in pulmonary flow resistance occurred only when the mixture was administered at high relative humidity.
		NaCl aerosol	1.00	-	N/A	
Amdur and Corn (1963)	Guinea pig	SO_2 +	6.5	-	N/A	This combination resulted in an increase in pulmonary flow resistance exceeding that produced by either compound alone.
		$ZnSO_4(NH_4)_2$-$SO_4 \cdot 6H_2O$	0.25	-	N/A	
Amdur (1978)	Guinea pig	SO_2 +	0.78	0.30	~1 hr	Statistically significant changes in resistance and compliance. Effect of $CuSO_4$ + SO_2 combination was more than additive.
		$(NH_4)_2SO_4$	0.5			
		SO_2 +	0.832	0.32	~1 hr	
		NH_4HSO_4	0.9			
		SO_2 +	0.936	0.36	~1 hr	
		$CuSO_4$	0.4			
		SO_2 +	0.806	0.31	~1 hr	
		Na_2SO_4 (salt particle size: 0.1 μm)	0.9			

Table A-10 (Continued). SUMMARY TABLE: EXPOSURE TO SULFUR DIOXIDE + PARTICULATES, AEROSOLS, AND H_2SO_4

Author	Species	Substance	Concentration mg/m^3	Concentration ppm	Duration	Response
Amdur (1978)	Guinea pig	SO_2	-	0.4-0.8	1-2 hr	No apparent synergism between the two gases under exposure conditions.
		+				
		O_3	-	0.4-0.8		
		SO_2	2.6-26	1-10		Increase in resistance.
		+				
		Oil mists	10	-		

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A.4 CLINICAL STUDIES

In attempting to relate dose-response effects in the laboratory to those occurring under ambient conditions, many parameters must be considered. For instance, it should be noted that a low dose causing some subjective discomfort or after-effect in an individual may not be measurable in the clinical setting. On the other hand, a slight effect measured in the upper airways during a clinical exposure may go unnoticed by the experimental subject. Such an effect would also go unnoticed by an individual exposed under ambient conditions.

Of the sulfur compounds emitted from mobile sources, sulfur dioxide and sulfuric acid have received the most clinical study because of their abundance in the ambient air and in the workplace. Less research has been done on sulfate, hydrogen sulfide, and carbonyl sulfide. Because the latter two compounds occur at relatively small concentrations in the ambient air--they form sulfur oxides fairly rapidly in the presence of oxygen--they are not considered as potentially hazardous mobile source emissions. Suspended sulfates, however, not only are potentially hazardous, but have been demonstrated in some animal and human studies to be the most toxic of the sulfur compounds discussed.

Sulfur dioxide concentrations of about 1-5 ppm, levels which exist during air pollution episodes, have been found to exert measurable effects on lung function in the laboratory. Clinical tests indicate that some individuals are more susceptible than others to the effects of SO_2. Within the concentrations tested, however, the effects of this pollutant have been transitory.

Studies of sulfuric acid show effects occurring at 1.0 mg/m^3. These effects, revealed by an increase in respiratory rate, have been found to be partially dependent on the size of the particles in the mist. Clinical studies of pollutant combinations have, like some animal experiments, suggested the existence of a synergistic effect under certain conditions.

A.4.1 Sulfur Dioxide

Pulmonary mechanics can be altered in human subjects exposed briefly to sulfur dioxide. Frank et al. (1962) exposed 11 subjects for 10 min to three concentrations of the gas (1, 5, and 13 ppm) and observed concentration-related effects on pulmonary flow resistance. The low concentration produced an increase in resistance in one subject, while the high concentration produced an increase in all subjects. The intermediate concentration was associated with a 39 percent increase in the flow resistance of the group. Under the 10-min exposure schedule, the observed effects appeared within 1 min and peaked within 5 min of the beginning of the period. When the exposure period was lengthened to 30 min, the percent increase in flow resistance was less than that noted after the 10-min exposure.

Weir and Bromberg (1972, 1973) exposed 19 males to three concentrations of SO_2 (0.3, 1.0, and 3.0 ppm) for 96 and 120 hr. Twelve of the subjects were in a normal state of health, while the other seven were smokers with early symptoms of chronic pulmonary disease. The seven smokers exhibited no significant increase in pulmonary flow resistance in response to any of the test exposures. The 12 healthy subjects, however, showed significant increases in airway resistance at the high exposure concentration, an effect which disappeared within 48 hr after the end of the exposure. These findings led the investigators to conclude that persons with preexisting peripheral airway disease are not significantly more susceptible to SO_2 effects than are healthy persons.

Speizer and Frank (1965) ran a series of experiments to compare the effects of sulfur dioxide (15 and 28 ppm for 10 min) when breathed in by the mouth and when breathed in by the nose. They found that breathing SO_2 by mouth increased flow resistance in 9 of 12 experiments, while breathing SO_2 by nose increased resistance in 3 of 12 experiments. The data indicated that less than 1 percent of the SO_2 entering the nose reached the oropharynx.

A subsequent study by Anderson et al. (1974) examined the effects of nasal breathing of sulfur dioxide on lung mechanics, mucus flow rate, and subject discomfort. Fifteen young male subjects were exposed for 6 hr to SO_2 concentrations of 1, 5, and 25 ppm. At the two higher concentrations, the subjects exhibited a significant decrease in nasal flow rate, particularly in the anterior nose. This effect was especially marked in individuals with initially slow mucus flow rates. In support of previous studies indicating that the nose acts as an SO_2 scrubber, the investigators found that pharyngeal air samples from the subjects contained less than 1 percent of the total SO_2 inhaled. Although changes were noted in nasal flow resistance and forced expiratory volume, the closing volumes, which will reflect changes at the smaller airways, were not affected by the two higher concentrations. The 1-ppm concentrations, however, caused a general constriction in the upper airways. Finally, dryness and/or slight pain in the nose and pharynx occurred at the higher doses. Subject ratings of discomfort were proportional to the exposure concentraton.

Before the study by Anderson et al., Cralley (1942) investigated the effects on mucus flow and ciliary action of short-term SO_2 exposures. He noted a small decrease in the rate of mucus removal after a 60-min exposure to 10-15 ppm. A nearly 50 percent decrease in ciliary activity was noted with a 30- to 60-min exposure to 25 ppm. When a 30-min treatment of 50 ppm was administered, a nearly 70 percent reduction in ciliary activity was observed. Considered within the limits and conditions of the test design, these effects were judged to be dose related.

Because mucus traps microbes and other particles inhaled from the ambient air and participates in pulmonary clearance, a reduction in its flow rate could increase susceptibility to infectious disease. To test this hypothesis, Anderson et al. (1977) inoculated 32 subjects with rhinovirus type 3 and exposed half the group to 5 ppm SO_2 for 4 hr. Although a 50 percent decrease in mucus flow rate occurred in the subjects exposed to SO_2, there was no increase in the number of colds they developed. In fact, the investigators noted that the SO_2-exposed subjects had fewer symptoms and a more persistent, but lower, level of virus proliferation.

Newhouse et al. (1978) assessed bronchial clearance and pulmonary function in healthy nonsmoking males and females exposed via the mouth to 5 ppm SO_2 for up to 2-1/2 hr. The subjects exercised strenuously and periodically during the exposure (the heart rate remained at 70-75 percent of the predicted maximum). In addition, the clearance of a radioactive aerosol (3-μm mean mass diameter [MMD]), deposited before SO_2 exposure, was measured. After a 2-hr exposure to SO_2, the clearance of the inhaled particles increased, accompanied by a decrease in the maximum mid-expiratory flow rates. No significant effects on vital capacity or forced expiratory volume were observed.

Lawther et al. (1975) conducted a 4-year study with 25 healthy male and female subjects to examine the effect of sulfur dioxide on pulmonary function. They used the product of two parameters (airway resistance and thoracic gas volume) to describe specific airway resistance (SR_{aw}). Reaffirming much of the previously reported research, they found an increase in SR_{aw} in mouth-breathing subjects exposed to 1-3 ppm SO_2. In one study, an increase in this value was observed after only eight deep breaths of a 3-ppm concentration. Changes in ventilatory function occurred throughout the exposure schemes, but these changes were transient and varied among the subjects.

Jaeger and Wittig (1978) studied SO_2 effects in normal subjects and in asthmatic patients. Among the 40 normal subjects, an exposure of 0.5 ppm SO_2 produced pulmonary changes in only one individual--a transient episode of wheezing dyspnea. A similar exposure produced slight changes in several of the 40 asthmatics. In terms of group averages, mid-expiratory flow rates were 3 percent lower in the asthmatic subjects than in the normal subjects. Two of the asthmatics experienced wheezing dyspnea. (Since fumes from a kerosene space heater were sometimes present in the chamber, it is difficult to interpret the relative role of SO_2 in this study. However, in a study by Salem and Cullumbine [1961], kerosene smoke did not potentiate the effects of SO_2 or H_2SO_4 in guinea pigs or mice.)

Snashall and Lewin (1976) examined, among other things, the inhibitory effects of atropine and sodium cromoglycate (SCG) on bronchial response in normal and asthmatic subjects exposed to SO_2. (The drug atropine will block some vagally mediated effects, while SCG prevents the release of histamine and other vasoactive substances from mast cells.) As expected, a 3-min exposure to 3 ppm SO_2 increased airway resistance in all subjects. However, atropine did not inhibit the respiratory hyperactivity associated with SO_2 exposure in either normal or asthmatic subjects. This lack of inhibition led the researchers to conclude that vagal mechanisms play little role in causing hyperreactivity associated with sulfur dioxide.

A.4.2 Sulfuric Acid and Sulfates

As noted in the discussion of animal in vivo studies, sulfuric acid is more irritating (exerts greater effects on flow resistance and respiratory rate) than sulfur dioxide. Its action varies according to relative humidity, aerosol particle size, and individual response, but studies generally have shown that H_2SO_4 increases airway resistance at about a 1-mg/m^3 concentration. Relatively little research has focused on the effects of smaller concentrations on human subjects. In evaluating the effects of sulfuric acid emissions, it should be noted that H_2SO_4 reacts in the atmosphere to form various sulfate salts.

Amdur et al. (1952) exposed 15 normal human subjects to 0.35- to 5.0-mg/m^3 sulfuric acid concentrations for 5 to 15 min. The mist caused changes in respiration at all test concentrations. Between 0.35- and 0.5-mg/m^3 concentrations, the respiration rate increased by about 30 percent, coinciding with a 28 percent decrease in deep breathing. Similar findings were reported by Bushtueva (1957) and Morando (1956). This change may have been a reflex response to protect against the retention of the acid particles.

The effects of a 2-hr exposure to 1 mg/m^3 H_2SO_4 (0.5-μm MMD) on bronchial clearance and pulmonary function were investigated by Newhouse et al. (1978).

The 10 subjects were healthy, male and female nonsmokers who exercised during the exposure. In addition, prior to exposure, all subjects inhaled radiolabeled particles (3-μm MMD). After the exposure, five of the subjects had a marked increase in clearance of the particles. There was no significant alteration of vital capacity or forced expiratory volume. However, there was a marginally significant decrease of 1.4 percent ($P = 0.09$) in the maximum mid-expiratory flow rate.

Recently, Larson et al. (1977) considered the presence of ammonia in the upper airways as a neutralizer of sulfuric acid. On the basis of a diffusion calculation, they noted that at 30 percent relative humidity, a 20-$\mu g/m^3$ concentration of sulfuric acid mist of 0.3-μm particles should be completely neutralized after about 0.5 sec in the nose and 0.1 sec in the mouth. This calculation suggests that the mouth is more efficient than the nose in neutralizing acid aerosol.

Although a number of animal studies have considered the effects of exposure to sulfate salts, which are present during air pollution episodes, relatively little research has been reported for humans. Animal studies have demonstrated that the toxicity of these salts is influenced by particle size and the metal ions present in them. One of these salts, zinc ammonium sulfate, which has been demonstrated as a respiratory irritant in animals, was reported as a constituent of the Donora, Pennsylvania, pollution episode of 1948.

Hackney et al. (1978) exposed normal, sensitive, and asthmatic subjects for 2-1/2 hr daily over several successive days to 100 $\mu g/m^3$ ammonium sulfate, 85 $\mu g/m^3$ ammonium bisulfate, or 75 $\mu g/m^3$ sulfuric acid aerosol. The particle size of all three compounds was nominally 0.3 μm (MMD). The exposure concentrations were chosen on the basis of the highest reported sulfate ion concentrations detected in ambient air pollution episodes in the Los Angeles Basin. During the test exposure intervals, in which the chamber temperature was maintained at 31°C, the subjects engaged in intermittent light exercise to simulate typical outdoor activity such as might occur during a summer day pollution episode. In general, the investigators found little or no evidence of health effects from these exposures in terms of changes in various respiratory measurements. These studies failed to confirm the association between sulfates and morbidity often observed in epidemiological studies. According to the investigators, one possible explanation is that sulfates produce adverse health effects through additive or synergistic interaction with coexisting pollutants.

A.4.3 Pollutant Interactions

In 1964, Frank et al. reported a study comparing the effects of SO_2 alone and in combination with sodium chloride (NaCl). Healthy adults were exposed to 1-2, 4-6, or 14-17 ppm SO_2 for 30 min in the presence or absence of an NaCl aerosol of submicron particles. No systemic differences were found between the gas and gas-aerosol exposures. Basically, the same dose-response relationship existed under both circumstances. As noted in the *in vivo* section, when Amdur (1959) exposed guinea pigs to this combination, it produced greater changes in pulmonary mechanics than did SO_2 alone. This difference in species response has not been explained.

Toyama (1962) exposed 13 subjects to SO_2 with and without NaCl aerosol. After initial control measurements, the subjects were exposed sequentially for 5 min to NaCl (0.22-µm count median diameter [CMD]), SO_2 (1.6-56 ppm), and an SO_2-NaCl combination. An increase in flow resistance occurred after exposure to SO_2. When SO_2 was combined with NaCl aerosol, the increase in flow resistance was greater. Similar results were reported by Nakamura in 1964.

Using lower concentrations of the same compounds, Burton et al. (1969) exposed 10 healthy adult males to 2-2.7 mg/m^3 NaCl (1-µm CMD) and 1-3 ppm SO_2. Neither SO_2 alone nor combined with the NaCl aerosol altered pulmonary mechanics. At the concentrations used, this result correlates with data based on animal studies by Amdur (1960).

Hazucha and Bates (1975) measured the effect of a combination of SO_2 and ozone on pulmonary function. Eight healthy young adults were exposed to 0.37 ppm ozone and/or 0.37 ppm sulfur dioxide for 2 hr. While the extent of ventilatory function effects varied considerably among the individual subjects, it was evident that these changes were greater for the pollutant combination than for either pollutant alone. The combination decreased forced expiratory volume by 22 percent, peak expiratory flow rate by 21 percent, and vital capacity by 8 percent.

A similar study by Bell et al. (1975, 1977) examined the effects of the same pollutant concentrations on eight Los Angeles residents, four of whom had respiratory conditions which rendered them sensitive to changes in air quality. In this study, the effect of acute exposure to the combination was greater than that exerted by O_3 alone, but the magnitude of response was considerably less than that reported by Hazucha and Bates.

Horvath and Folinsbee (1977) exposed young human males to SO_2 and O_3 singly and combined under one environmental condition--25°C and 45 percent relative humidity. All nine subjects had 15-min intervals of walking and resting during a 2-hr exposure scheme. The subjects exposed to filtered air or 0.40 ppm SO_2 showed no significant changes in pulmonary functions. Those exposed to 0.40 ppm O_3 singly or combined with 0.40 ppm SO_2 had significant decreases in maximum expiratory flow, forced vital capacity, and inspiratory capacity. However, these decreased pulmonary functions were attributed to O_3 alone and not to the pollutant combination. One of the subjects had an enhanced sensitivity to the O_3-SO_2 combination. This general absence of synergistic effect correlates with the Amdur study (1978) showing no synergistic effect in guinea pigs exposed to the SO_2-O_3 combination.

von Nieding and co-workers (1977) exposed nine human volunteers to combined NO_2, O_3, and SO_2 at up to 5, 1, and 5 ppm, respectively. They found significant increases in airway resistance and reductions in arterial oxygen partial pressure. The PO_2 returned to normal values 1 hr following the termination of exposure, while the airway resistance remained slightly elevated. In addition, bronchial challenge with an aerosol containing 1-3 percent acetylcholine (a bronchoconstricting agent) produced an increase in bronchial reaction following exposure to the combined gases.

In conclusion, clinical studies show that most human subjects respond to 5 ppm (13,000 $\mu g/m^3$) SO_2, while certain sensitive subjects respond to 1-2 ppm (2600-5200 $\mu g/m^3$) SO_2. The typical response is an increase in pulmonary flow resistance, although at lower levels, e.g., 1 ppm SO_2 (2600 $\mu g/m^3$), significant decreases in nasal mucus flow rate have been demonstrated. Unlike H_2SO_4, SO_2 is removed more efficiently through nose breathing than through mouth breathing.

The effects of H_2SO_4 on human subjects involve primarily changes in respiration rate and bronchoconstriction at concentrations of 0.35-5.0 $\mu g/m^3$ for 5-15 min. The presence of ammonia in the upper airways seems to mitigate some effects of H_2SO_4 through neutralization. Nevertheless, the percent relative humidity and particle size seem to determine the relative toxicity of sulfate aerosols.

Finally, recent animal and human studies indicate that SO_2 may not have synergistic activity in combination with O_3. A summary of some human responses to sulfur-bearing compounds is given in Table A-11.

Table A-11. SUMMARY TABLE: EFFECTS OF SULFUR IN MAN

Chemical	Concentration, mg/m^3	Time	Effect	Reference
SO_2	0.96-1.8	20 sec-15 min	Decreased alpha wave in EEG, increased light sensitivity	Bushtueva et al. (1962) Dubrovskaya (1960)
SO_2	0.78-15.6	Up to 120 hr continuous	Changed S-sulfonate plasma levels	Gunnison and Palmes (1974)
SO_2	2.6, 13, 33	10-30 min	Increased pulmonary resistance (dose-response effects)	Frank et al. (1962)
$SO_2 + NO_2$	13 + 10.3	2 hr	Decreased arterial O_2 tension, increased pulmonary resistance	von Nieding et al. (1977)
$SO_2 + H_2SO_4$	0.9 + 0.6	3-15 min	Changed optical response	Bushtueva (1961)
$SO_2 + O_3$	1.05 + 0.72	2 hr	Decreased pulmonary function	Hazucha and Bates (1975)
$SO_2 + SO_3$	1.0 + 0.8	2 hr	Decreased pulmonary function attributed to O_3 alone	Horvath and Folinsbee (1977)
SO_2 + NaCl	4.6 + 160	5 min	Increased pulmonary resistance	Toyama (1962)
H_2SO_4	0.35-5.0	5-15 min	Reflex bronchoconstriction, shallow rapid breathing, and decreased minute ventilation	Amdur et al. (1952)
H_2SO_4	0.6-2.4	60-120 min	Transient increased light sensitivity	Bushtueva (1960, 1962)

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A.5 EPIDEMIOLOGY

As compared with laboratory studies, the epidemiological approach to investigating air pollutant effects involves considering many variables over long periods of time. However, when designed and executed properly, these studies provide very useful information. Moreover, epidemiological surveys are necessary to ascertain the minimal concentrations of air pollutants that produce deleterious health effects in human populations. For example, information derived in part through epidemiological investigation formed the basis for Federal air quality standards. The current primary standard for sulfur dioxide, considered as the maximum limit, beyond which health effects may occur, states that concentrations averaged over a given 24-hr period may not exceed 365 $\mu g/m^3$ and must be held to an annual arithmetic mean of 80 $\mu g/m^3$.

To date, epidemiological studies on ambient sulfur compounds have focused primarily on stationary sources, which account for about 98 percent of sulfur emissions. Nevertheless, certain inferences regarding emissions from mobile sources may be made from the information these studies have provided. Table A-12 summarizes data from several studies which have linked pollution with human mortality and morbidity. Included are pollution episodes and their effects on the general population, as well as on especially sensitive groups such as cardiopulmonary patients, bronchitis patients, and children with asthma. While these studies are of little direct help in determining the health consequences of mobile source emissions, they do help to establish conditions under which pollutant concentrations are likely to be hazardous.

Table A-12. SUMMARY TABLE: MORBIDITY AND MORTALITY

		Exposure		
Author	Location/date	Substance	Concentration estimated	Observations
Firket (1931)	Meuse Valley, Belgium Dec. 1-5, 1930	SO_2	Peaked at 25.20 mg/m³ (9 ppm)[a]	63 deaths. Many people ill with respiratory symptoms.
Schrenk et al. (1949)	Donora, Pa. Feb.-April 1949	SO_2	0.0-1.586 mg/m³ (0.0-0.61 ppm)[a]	While no single substance was responsible for the Oct. 1948 episode, the occurrence could have been produced by a combination of two or more contaminants. SO_2 and its oxidation products together with particulate matter are considered significant contaminants.
		Total sulfur	0.0-2.184 mg/m³ (0.0-0.84 ppm)[a]	
		Particulates	~0.0-5.32 mg/m³	
		Zinc	~0.0-0.0327 mg/m³	
		Lead	~0.0-0.67 mg/m³	
Wilkins (1954)	London Dec. 5-9, 1952	Smoke	1.41 mg/m³	Daily number of deaths during the smog incident followed the sharp upward trend of SO_2 and smoke concentrations. Time lag between cause and initial effect was approximately 12 hr. Excess mortality equaled 4000 deaths.
		SO_2	1.482 mg/m³/day (0.57 ppm)[a]	
Bradley et al. (1958)	London Dec. 2-5, 1957	Black suspended matter	~0.84-2.33 mg/m³	Fog estimated to have caused between 800 and 1000 deaths in Greater London. Meteorologically it was not as consistently dense nor were the levels of pollution in most areas as high as previous in fog episodes known to be associated with a similar high mortality.
		SO_2	624-1638 μg/m³/day (~0.24-0.63 ppm)[a]	

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[a] Originally reported as ppm. Conversion factor 1 ppm = 2600 μg/m³ was used.

Table A-12 (Continued). SUMMARY TABLE: MORBIDITY AND MORTALITY

		Exposure		
Author	Location/date	Substance	Concentration estimated	Observations
Greenburg et al. (1962)	New York City Nov. 1953	SO_2	2236 μg/m³/day (0.86 ppm)[a] (max. 1/2-hr value)	Excess deaths related to elevated concentrations of SO_2 and TSP.
			390-520 μg/m³/day (0.15-0.2 ppm)[a] (avg daily value)	
		Smoke	2.5-6 COHS	
Buechley et al. (1973)	New York-New Jersey-Connecticut 1962-1966	SO_2	~0.02-1.30 mg/m³/day	Analysis of deaths for New York metropolitan region with daily SO measurements showed that the pollution measurement contributes significantly to prediction of deaths. Mortality was 1.5% less than expected on 232 days with SO_2 levels below 0.03 mg/m³ and 20% greater than expected on 260 days with SO_2 levels above 0.5 mg/m³.
		COHS	Not specified	COHS does as well in predicting deaths.

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[a] Originally reported as ppm. Conversion factor 1 ppm = 2600 μg/m³ was used.

Table A-12 (Continued). SUMMARY TABLE: MORBIDITY AND MORTALITY

Author	Location/date	Exposure		Observations
		Substance	Concentration estimated	
Schimmel and Greenburg (1972)	New York City Jan. 1963-Dec. 1968	SO_2	Mean: 448.5 μg/m³/day (0.1725 ppm)[a] (SD: 0.08 ppm)[a]	Estimated average daily excess deaths ranged from 18.2 to 36.74, with an intermediate estimate of 28.63. This latter figure would represent about 10,000 deaths a year which would not have occurred at the time they did if there had been no pollution on the day of death or on immediately preceding days. This represents 12% of the over 1/2 million deaths occurring during the 6-year period.
		Smoke shade	Mean: 2.103 COHS SD: 0.702 (fully adjusted)	
			Mean temp: 54.41°F SD: 17.46°F	
				Over 40% of the respiratory disease deaths were associated with short-term pollution effects.
McCarroll and Bradley (1966)b	New York City Dec. 1962	COH SO_2	6 2.1 mg/m³/day	Excess deaths in 45-64 age group and over-65 age group.
	Jan. 1963	COH SO_2	6 1.7 mg/m³/day	Peak in death rate superimposed on high deaths due to influenza.
	1962-1964			Some periods of high air pollution appeared to be related to air pollution; relationship was not clear during other periods of high air pollution.

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[a] Originally reported as ppm. Conversion factor 1 ppm = 2600 μg/m³ was used.

[b] Information from Air Pollution: Air Quality Criteria for Sulfur Oxides.

Table A-12 (Continued). SUMMARY TABLE: MORBIDITY AND MORTALITY

		Exposure		
Author	Location/date	Substance	Concentration estimated	Observations
Becker et al. (1968)	New York City Metropolitan area Nov. 23-27, 1966		24-hr averages	
		API	~3-34	Increased symptoms of dyspnea, cough, sputum, wheeze, eye irritation, and general discomfort coincided with increased pollutant levels.
		Smoke shade (COHS)	~3-6	
		CO_2	~3-13	
		SO_2	~6.5 mg/m^3 (0.2-0.5 ppm)[a]	Subjects with prior respiratory + COPD disease were significantly more affected than those without ($P \leq 0.05$).
				No significant difference between symptom responses of smokers and nonsmokers was found.
				Subjects smoking <10 cigarettes/day reported an increase in coughing; >20 cigarettes/day had increased difficulty in breathing; 10-20 cigarettes/day had the least symptom response.

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[a] Originally reported as ppm. Conversion factor 1 ppm = 2600 $\mu g/m^3$ was used.

Table A-12 (Continued). SUMMARY TABLE: MORBIDITY AND MORTALITY

		Exposure		
Author	Location/date	Substance	Concentration estimated	Observations
McCarroll (1967)	New York City		24-hr averages	
	January 1962	Smoke SO_2	0.80 mg/m^3 1.45 mg/m^3	50 excess deaths in 1 day.
	February 1962	Smoke SO_2	0.60 mg/m^3 1.45 mg/m^3	40 excess deaths over a 2-day period.
	December 1962	Smoke SO_2	0.80 mg/m^3 2.00 mg/m^3	90 excess deaths over a 2-day period following a peak pollution episode.
Douglas and Waller (1966)	Various locations in Great Britain 1946-1965	SO_2 Smoke	>0.13 mg/m^3 >0.13 mg/m^3	Among school children, SO_2 and smoke at the indicated concentrations were associated with increased frequency and severity of lower respiratory diseases. Upper respiratory diseases were not related to pollution concentrations.

Table A-12 (Continued). SUMMARY TABLE: MORBIDITY AND MORTALITY

		Exposure		
Author	Location/date	Substance	Concentration estimated	Observations
Sterling et al. (1967)	Los Angeles, Calif. March 17-Oct. 26, 1961		Avg value for 10 successive days	Apparent correlation between length of stay in hospital and pollutants SO_2 ($P \leq 0.01$), total oxides of nitrogen ($P \leq 0.01$), NO_2 ($P \leq 0.005$), and particulate matter ($P \leq 0.01$).
		Oxidant	0.0368 ppm	
		CO	9.53 ppm	
		SO_2	33.5 μg/m^3 (0.0129 ppm)^a	
		NO_2	0.0624 ppm	Consistent and significant negative correlation between O_3 and "relevant" and heart disease categories. (Effect may be mediated by temperature and humidity.) For total relevant diseases, correlations are significant for SO_2, NO_2, total NOx, O_3, and particulate matter.
		NO	0.0553 ppm	
		NOx (total)	0.119 ppm	
		O_3	0.418 ppm	
		Oxidant precursors	0.0275 ppm	
		Particulates	30.1 Km units x 10	
Goldberg et al. (1974)	New York City 1950's	TSP, including sulfate	Measured weekly	A study was made of patients with bronchopulmonary symptoms, mainly elderly people, to determine the effects of pollution on respiratory response. Sulfate was considered to show the strongest effect on asthma attack rates, but estimates of 50-70 μg/m^3 were made for the threshold of effects of TSP.

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[a] Originally reported as ppm. Conversion factor 1 ppm = 2600 μg/m^3 was used.

At present, it is virtually impossible to sort out the roles of the various sulfur compounds exerting effects on health. Acute pollution episodes are characterized by high levels of sulfur dioxide and particles, along with high humidity and weather conditions which prevent dispersion of contaminants. Most studies have not isolated sulfate from other types of suspended particles. These unknowns make it difficult to establish emissions/health relationships in specific terms.

In assessing a health hazard, a number of factors should be considered, such as the duration and concentration of exposure, the level of activity, and preexisting illness or sensitivity (e.g., pregnancy). For example, it should be remembered that a mean ambient concentration is often less significant than the variations around the mean. Thus, a short-term peak within a 24-hr mean concentration could have more effect than a low, long-term exposure. Another consideration is the level of activity of individuals. Children in strenuous play would in effect have more exposure to ambient pollutants than the elderly in retirement. In addition, functional and structural dimensions of the respiratory system also determine dose response. Thus, preexisting disease in the airway can affect the distribution and clearance of particles; some regions could become "hot spots."

In reviewing the epidemiological literature, it becomes evident that acute pollution episodes with high sulfur dioxide and total suspended particle (TSP) levels may lead to fatalities within the exposed population. The fog which occurred in the Meuse Valley, Belgium, in 1930 was associated with 60 excess deaths in a small community. The levels of atmospheric pollutants, particularly sulfur dioxide, though not measured, were thought to be very high

(Firket 1931). A similar incident occurred in Donora, Pennsylvania, in 1948, causing 20 deaths and acute illness in about 6000 individuals. In all, nearly half the population of 13,000 was affected (Schrenk et al. 1949).

These are striking examples of air pollution disasters, but the specific pollutant concentrations occurring during these episodes were not quantitatively measured. Other studies of mortality have considered long-term, low-level exposure as opposed to episodic exposure. For example, Buechley et al. (1973) studied mortality patterns and pollution levels over a 4-year period in the New York metropolitan region and found that death rates correlated with SO_2 concentrations. Taking into account other variables affecting mortality (such as extreme weather conditions), they determined that mortality was 1.5 percent less than average on days when SO_2 levels were below 0.03 mg/m^3 and 20 percent higher than average on days when SO_2 levels exceeded 0.5 mg/m^3.

In London, a 4-day "killer" fog occurred in December 1952, during which time smoke levels reached nearly 4.5 mg/m^3 and sulfur dioxide levels reached 2.8 mg/m^3 (48-hr average). Studies have estimated that 4000 deaths resulted from this exposure (for example, Logan 1953). The death rate began to rise within 24 hr of the beginning of the pollution episode and fell abruptly to near-normal levels when the fog abated. Most deaths occurred among people with preexisting disease, including bronchitis (tenfold increase in deaths) and coronary heart disease (threefold increase).

Studies of this episode reported an increase of nearly 50 percent in admissions to hospitals in Greater London, due almost entirely to patients admitted with respiratory disease (Ministry of Health 1954). The effects on one doctor's general family practice in London (Fry 1953) during the week of the fog and the week following included twice as many cases of upper respiratory symptoms (sore throat, cough, nasal discharge) as usual and three times as many cases of lower respiratory disease. Of the 105 patients with known chronic respiratory disease, two died and another 35 were affected to some degree.

Studies of subsequent fog episodes in London have correlated daily mortality with daily levels of SO_2 and particles. Martin (1964) reviewed studies of several incidents of polluted fog in London after 1952, presenting his own results for the winters of 1958-59 and 1959-60. He measured the number of deaths from all causes and the number of applications for emergency medical service related to cardiovascular and respiratory conditions. He then matched these measures of mortality and morbidity each day with the levels of sulfur dioxide and particles on the same day. On days of high levels of either pollutant there were high levels of mortality and morbidity. The results are shown in Table A-13.

The effect of acute episodes of very high pollution is a dramatic increase in mortality and morbidity, predominantly among the elderly or those with chronic preexisting disease. The effect of exposure to more moderate levels of sulfur dioxide and other sulfur compounds over longer periods of time has also been examined. Studies have been of several types: surveys of populations with different exposure levels, longitudinal studies of populations in the same geographic area, and comparisons over time of populations in different geographic areas. The variables used to investigate the presumed effects of sulfur compounds have included measures of respiratory function, Table A-13. MORTALITY AND MORBIDITY ON DAYS WITH HIGH SO_2 LEVELS OR HIGH SMOKE LEVELS

Range of values, mg/m^3	No. of days	Mean excess of mortality	Mean excess of morbidity
		SO_2	
0.9-1.28	5	39.6	12.8
0.8-0.89	6	19.2	12.3
0.7-0.79	4	29.8	9.8
0.6-0.69	5	4.4	7.0
0.5-0.59	6	11.6	5.2
0.4-0.49	9	9.0	2.2
		Smoke	
1.1-1.51	7	40.0	12.9
1.0-1.09	4	13.8	4.8
0.9-0.99	4	17.5	5.0
0.8-0.89	0	-	-
0.7-0.79	9	9.2	2.4
0.6-0.69	6	13.0	-0.7
0.5-0.59	9	5.2	3.2

Source: Martin (1964).

the prevalence of respiratory disease, and the incidence and course of acute disease. There is a large body of evidence relating SO_2 and chronic bronchitis, but the exact mechanism of the relationship is unknown. Further long-term population studies are needed, along with animal studies, to investigate the role of SO_2 in the onset, prevalence, and course of chronic bronchitis.

Among adults without preexisting illness, the relationship between acute respiratory disease and SO_2 is more tenuous than it is in children. Epidemiological studies regarding community and industrial exposures have produced inconsistent results, subject to interpretation. Limited experimental studies have not shown increased rates or severity of respiratory infection resulting from SO_2 exposure, although it is generally conceded that data gathered under controlled conditions may not reflect conditions measured epidemiologically.

Pulmonary function has been the most extensively studied biologic response to SO_2, primarily because it is a convenient, noninvasive technique. Evidence from several lines of investigation, including epidemiological studies, has supported laboratory studies showing that SO_2 impairs pulmonary function by increasing airway resistance. The link between this altered pulmonary function and subsequent disease states is still unknown.

Sulfur oxide exposure and respiratory morbidity in children have recently been reviewed by Hammer (1977). Studies from several countries clearly associate excess lower respiratory tract morbidity in the young with exposure to sulfur oxides. For example, Douglas and Waller (1966) conducted a long-term study of British school children and found increased frequency and severity of lower respiratory diseases in association with annual mean smoke concentrations above 130 $\mu g/m^3$ and sulfur dioxide levels above 130 $\mu g/m^3$. Both acute and chronic upper respiratory disease in children have also been linked to sulfur oxide exposure. Lunn et al. (1967) studied 819 school children in Sheffield, England, and found that both upper and lower respiratory tract infections were related to actual air pollution measurements. The mean daily averages measured at a single station were 300 $\mu g/m^3$ for smoke and 275 $\mu g/m^3$ for SO_2. Several studies have found that pulmonary function in children varies inversely with pollution exposure (Hammer 1977).

Relatively few studies have been concerned with air pollution and asthmatic children per se, and there is little information available on the prevalence of childhood asthma in communities with high and low pollution levels. No consistent trends of asthma prevalence and air pollution were found in three surveys of American studies (Hammer 1977). A recent report from Japan did find an increased prevalence of asthma and related allergic conditions in a high-pollution community which apparently had exposure to petrochemical wastes as well as sulfur dioxide and total suspended particulate matter (Yoshida 1976).

As was pointed out earlier, one of the problems associated with the epidemiological study of air pollution effects is the difficulty of sorting out the specific roles of the various compounds emitted. Especially elusive are the effects of sulfuric acid aerosol, a mobile source contaminant of concern. The problem is that the technology is just now becoming available for ambient measurements of this aerosol on a community-wide scale. Some studies have been conducted on exposure in industrial settings where concentrations far exceed ambient concentrations. For example, Williams (1970) reported a 12-year study of sickness absence and pulmonary function in a group of English factory workers exposed to sulfuric acid aerosol. He found that workers exposed to high levels of aerosol suffered more spells of absence due to influenza, other upper respiratory tract disease, and bronchitis than did workers exposed to low levels of aerosol. Sulfuric acid aerosol concentrations were not monitored over the course of the study. However, a 1-day independent measure made in a high-exposure area of the factory showed concentrations varying between 3.0 and 16.6 mg/m^3, levels which so far exceed the probable limit of ambient concentrations as to render the results of this study valueless in terms of community epidemiology.

Rall (1974) reviewed the health effects of sulfur oxides and concluded that air pollution is caused by a complex mixture of chemical substances of varying toxicity of which the sulfur oxides are a principal component (see Table A-14). He recognized that the components which pose the primary hazards to human health and their respective contributions to human diseases have not been fully established as yet and that this is a source of some difficulty and controversy. However, there was a considerable body of evidence suggesting that there may be discernible human health effects from exposure to concentrations of SO_2 in the ambient air at levels close to current Federal primary standards.

Between 1967 and 1975, a series of studies was conducted to assess health effects from air pollutants. The U.S. Environmental Protection Agency formally titled a portion of these studies Community Health and Environmental Surveillance System (CHESS). One of the goals of CHESS

Table A-14. SUMMARY OF THE HEALTH EFFECTS FROM THE COMBINATION OF PARTICLES AND SO_2

Averaging time for pollution measurements	Place	Approximate levels of pollution: Particles, μg/m³	Approximate levels of pollution: SO_2, μg/m³	Effect
24 hr	London	2000	1144	Mortality
24 hr	London	750	700	Mortality
24 hr	London	300	600	Deterioration of patients
Weekly mean	London	200	400	Prevalence or incidence of respiratory illnesses
24 hr	New York	6[a]	1500	Mortality
Winter mean	Britain	100-200	100-200	Incapacity for work from bronchitis
Annual	Britain	70	90	Lower respiratory infections in children
	Britain	100	100	Upper and lower respiratory infections in children
	Britain	100	100	Bronchitis prevalence
	Britain	100	100	Prevalence of symptoms
	Buffalo	100	300[b]	Respiratory mortality
	Berlin, N.H.	180	731[b]	Increased respiratory symptoms Decreased pulmonary function

[a] In coefficient of base units (COHS).
[b] As μg of $SO_2/100\ cm^2$/day.

Source: Rall (1974).

was to provide a sound basis for relating human health effects to sulfur oxides and sulfates and to provide a reliable record of improvement in health for the U.S. population after reduction in air pollution.

Unfortunately, in the attempt to provide a large amount of data as quickly as possible, studies within the CHESS project showed defects in design, methods, and execution. Though generalizations from this group of studies were difficult, Finklea examined the body of data and presented best-judgment estimates of the threshold levels at which SO_2 and TSP may exert effects on health. In terms of annual concentrations, Finklea's estimate for threshold limits of SO_2 and particles was compatible with the results of earlier studies on which the Federal primary standards for these pollutants were based (80 $\mu g/m^3$). However, in terms of maximum 24-hr concentrations, his threshold estimate suggested that significant health effects occur at exposure levels well below the Federal primary standards for SO_2 and particles (365 and 260 $\mu g/m^3$, respectively). Questions regarding the validity of this finding have not been resolved. Nevertheless, the overall CHESS program does corroborate the notion that elevated air pollutants cause adverse health effects. But the precise relationship between sulfates and health effects cannot be quantified at present using data from this program.

To date, these and other epidemiological studies on sulfur compounds have focused primarily on stationary sources, which are responsible for 98 percent of the sulfur emissions. There are virtually no data showing effects on persons living near heavily traveled roadways, on automobile and airplane operators and passengers, or on bikers, joggers, and other pedestrians who could be exposed to the emissions. Nevertheless, data from the Los Angeles catalyst study provide a good empirical estimate of roadway exposure in one large metropolitan area. On the basis of nearly 2 years of sampling, average 24-hr freeway contributions of sulfur dioxide and suspended sulfate were estimated at about 12 and 1 $\mu g/m^3$, respectively.

In rare instances, sulfate emissions could amount to several micrograms per cubic meter and be associated with health effects. However, there are no sound epidemiological data to determine what the health effects might be. A cost/benefit analysis (Chapter 5) was initially performed for this report to assess those data. However, because of the difficulty in determining a threshold level at which health effects occur for sulfates, no real estimates could be made.

In conclusion, it appears that emissions of sulfur-bearing compounds from mobile sources at current emission rates pose no real hazard to the general population.

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APPENDIX B. WELFARE EFFECTS

B.1 VEGETATION

B.1.1 Introduction

This section reviews the effects of sulfur compounds on plant life. Sulfur dioxide (SO_2) is the most damaging of these compounds and is therefore reviewed in considerable detail. The effects of H_2S, H_2SO_4, and other sulfur compounds, though less documented, are also discussed.

Several early reports of damage from sulfur dioxide are of interest in that this pollutant was the first to be recognized for its phytotoxicity (Stoeckhardt 1871). Hartig (1896), Haselhoff and Lindau (1903), Sorauer (1904), Dana (1908), an Spaulding (1909) published various papers indicating that fume damage (most probabl SO_2) was responsible for injury to various crops. A small amount of research followed these initial studies until the 1920's, when the theory of "invisible injury" was proposed by Stoklasa (1923). Reports of sulfur dioxide-induced injury and its symptoms were scattered throughout the literature during the next two decades. Zimmerman and Crocker (1934), Whitby (1939), Thornton and Setterstrom (1940), and Katz (1949) added significantly to the accumulating information. Following the increased levels of emissions brought about by industrialization and associated power demands in the eastern portions of the United States and in Great Britain and Central Europe, reports of SO_2 injury to many forms of vegetation likewise dramatically increased. Recent advances in technology for monitoring various sulfur forms and analyzing plant tissue have once again spurred research in this area.

Several major topics of research have emerged in the past 10 years and will continue to be emphasized in the future:

1. Interaction of pollutants such as SO_2, ozone, nitrogen oxides, peroxyacytyl-nitrates, and halogens in combination.
2. Effects of long-term, low-level exposure.
3. Growth and yield effects observed in the field using air filtration systems.
4. Long-distance transport of polluted air masses and associated impacts of acidic rainfall at such distances.
5. Influence of the environment on the plant response.
6. Effects of sequential fumigations with single and multiple pollutants.
7. Pollutant and plant parasite interactions.

It should be noted that at this time there are no reported instances of vegetational injury induced by sulfur compounds generated by mobile (as distinct from stationary) sources. Recently, transportation-generated sulfate aerosol was thought to be a potential threat to vegetation. However, this possibility was refuted on the basis of ambient air quality data and information on vegetational dose-response relationships.

Nevertheless, the SO_2 emitted from mobile sources may be significant in causing plant injury when combined with other pollutants emitted into the urban environments. It is virtually impossible to separate out the combination effects of mobile versus point source SO_2 emissions once they have entered into gaseous mixtures with other pollutants.

Of final concern in making estimates of loss due to SO_2 emissions (or any other pollutant) is the concept of "hidden injury" as proposed by Stoklasa (1923). Such induced injury requires the measurement of some parameter, such as yield, that has been affected without the presence of visible symptoms. This concept has primarily been tested in relation to stationary sources of SO_2 emission. It remains to be thoroughly tested in relation to an urban environment where mobile and point sources emit numerous pollutants other than, or including, SO_2.

B.1.2 Chemistry

B.1.2.1 Forms of sulfur in the atmosphere and sulfur compounds affecting plant life--SO_2 constitutes the largest component of the gaseous sulfur compounds in the atmosphere and constitutes >95 percent of the sulfur compounds produced by the combustion of sulfur-containing fossil fuels. Moreover, most of the H_2S emitted into the atmosphere is rapidly oxidized to SO_2.

Sulfur dioxide is catalyzed by a variety of homogeneous and heterogeneous reactions to sulfates. One species of sulfate aerosol, sulfuric acid (H_2SO_4), is largely related to the major stationary sources of SO_2 emission. Transportation is not currently considered to be a major source for the acid aerosol, H_2S, or COS (U.S. Environmental Protection Agency 1977). Inorganic $SO_4^=$ aerosols also include $(NH_4)_2SO_4$, NH_4HSO_4, and others (Weiss et al. 1977).

In the coarse-particle range, sulfur is associated with both organic and inorganic particles and with the cations (Krupa 1978). These coarse particles consist of a combination of emissions, wind-blown dust, sea spray, volcanic products, and plant material (Whitby 1977) (Figure B-1).

Both dry and wet processes are involved in the deposition of gases, aerosols, and coarse particles to the ground. Dry fallout occurs most of the time, while wet

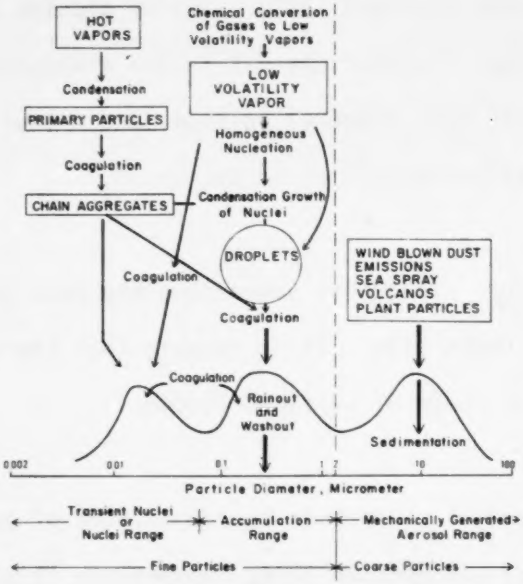
HOT VAPORS
Condensation
PRIMARY PARTICLES
Coagulation
CHAIN AGGREGATES
Chemical Conversion of Gases to Low Volatility Vapors
LOW VOLATILITY VAPOR
Homogeneous Nucleation
Condensation Growth of Nuclei
DROPLETS
Coagulation
Coagulation
WIND BLOWN DUST
EMISSIONS
SEA SPRAY
VOLCANOS
PLANT PARTICLES
Coagulation
Rainout and Washout
Sedimentation
0.002
0.01
0.1
1
2
10
100
Particle Diameter, Micrometer
Transient Nuclei or Nuclei Range
Accumulation Range
Mechanically Generated Aerosol Range
Fine Particles
Coarse Particles

Figure B-1. Schematic showing an atmospheric aerosol surface area distribution representing three modes, the main source of mass for each mode, the principal process involved in inserting mass into each mode, and the principal removal mechanisms.

Source: Whitby (1977).

fallout (rain and snow) depends on the frequency of precipitation and the geographic area. Acidic rain is one form of wet fallout that is of major concern. While the acidity of rain can be attributed to a great number of hydrogen ion donors in rain, many scientists believe that H_2SO_4 and HNO_3 are the major contributors (USDA Forest Service 1976).

B.1.2.2 Deposition rates of sulfur compounds--Deposition processes limit the lifetime of sulfur compounds in the atmosphere, the distance they travel, and their atmospheric concentrations (Garland 1977). Sulfur dioxide and particulate sulfate are the predominant forms of sulfur present in the atmosphere. Other atmospheric sulfur compounds, such as H_2S, dimethyl sulfide, and methyl mercaptan, are most likely deposited only after conversion to SO_2 or $SO_4^=$.

B.1.2.2.1 Dry deposition. Table B-1 summarizes the results obtained from several studies on SO_2 deposition. It is notable that the mean deposition velocities are similar for the wide range of surfaces listed.

There have been several studies of the deposition of particulate material on natural surfaces (Schnel and Hodgson 1974; Chamberlain 1975; Little and Wiffen 1977). Very large particles are chiefly deposited by sedimentation. Smaller particles, in the range of 1 to 100 μm, are borne by turbulence toward the surface, where sedimentation effects supplement their deposition. Submicron particles (H_2SO_4 aerosols) diffuse by Brownian motion through the thin laminar layers close to the surface elements, often followed by active uptake in the case of plant leaves.

Dry deposition can remove the larger particles from the atmosphere within 2 or 3 days, but this process would require several weeks to remove the submicron fraction.

Table B-1. RESULTS OF SEVERAL EXPERIMENTAL INVESTIGATIONS OF SO_2 DEPOSITION

		Mean		
		g	r_g	r_s
Surface	Method, etc.	scm^{-1}	scm^{-1}	scm^{-1}
Calcareous soil	Laboratory, mass balance			0.24-0.39
Acid soil	Laboratory, mass balance			1.2-3.8
Calcareous soil	Gradient, field	1.2	0.83	0.01
Grass	Gradient, field			
	Summer	0.8		0.8
	Autumn	0.3		3.0
Grass	$^{35}SO_2$ tracer, field	0.8		
Short grass	Gradient, field	0.85	1.2	0.34
Medium grass	Gradient, field	0.89	0.46	0.66
Medium grass	Tracer, field	0.19	0.38	0.45
Cotton sedgemoor	Gradient, field	0.7	-	-
Wheat	Gradient, field	0.74	-	1-2.5
Wheat		0.44	0.28	2.0
Soybean		1.25	0.11	0.69
Pine forest	Tracer	0.1-0.6	0.1	1.5-5.0
Pine forest	Tracer	1.0		

Notes: r_g = gas plus resistance, r_s = surface resistance; SO_2 deposition expressed in terms of deposition velocity relative to 1 m.

Source: Table summarized by Garland (1977).

B.1.2.2.2 Wet deposition. Both SO_2 and $SO_4^=$ can be dissolved in rain. Hogstrom (1973) found that sulfur emitted by a stationary source in Sweden was deposited by rain within 1 or 2 hr of its emission. However, Granat and Rodhe (1973) found that of the total amount of sulfur emitted from the tall stacks of a power-generating station, less than 6 percent was deposited by rain within a radius of 15 km. Davies (1976) measured the dissolved SO_2 in rainwater in England and found about 3 $mg/liter^{-1}$ when the corresponding air concentration was about 100 $\mu g/m^3$.

Garland (1977) summarized removal of sulfur from the atmosphere as follows:

1. SO_2 is removed by dry deposition at a deposition velocity of approximately 0.8 cm/sec^{-1} for most surfaces.
2. The deposition velocity of sulfate is no greater than 0.1 cm/sec^{-1}.
3. Sulfate is removed by rain with a time constant on the order of $10^{-4}/sec^{-1}$.
4. SO_2 removal by rain is approximately one order of magnitude less efficient.

B.1.2.3 Sulfur as a plant nutrient--Sulfur is a plant nutrient essential to the formation of amino acids. It occurs in plants as inorganic sulfate and as organically bonded sulfahydryl, disulfide, and sulfonic groups; it is also bonded in heterocyclic rings. The sulfahydryl form is important in producing the amino acids cysteine and methionine and in catalyzing coenzyme A. Organically bonded sulfur also contributes to the Calvin cycle of photosynthesis.

Inorganic sulfur occurs in plants as sulfate, which activates fermentation, maintains colloidal structure of protoplasm, increases assimilation activity (metabolism), and stimulates carbohydrate formation. Excess sulfate is stored in cell vacuoles as a sulfur reserve. Inorganic sulfate can exceed the level of organic sulfur by 5 to 10 times before injury occurs. Since the amount of stored sulfur has little effect on the level of organically bonded sulfur (Guderian 1970), the phytotoxicity can be attributed to inorganic sulfur.

The only positive effects of plant uptake of atmospheric sulfur dioxide occur areas with low-sulfur soils. Faller (1970) found that fumigating sulfur-deficient sunflower, corn, and tobacco plants with sulfur dioxide for several weeks promoted normal plant growth. Saalbach et al. (1962) found a slight increase in morrowstem yield by applying ammonium sulfate nitrate compared with lime nitrate or ammonium nitrate. Estimates show that 13 kg of sulfur per hectare (2.47 acres) was removed from soils by harvested products, similar to the amount of phosphorus thus removed, while the amount of sulfur removed through washout equaled 60 kg/ha. This yearly loss (73 kg/ha) was replaced by fertilizer (19 kg/ha) and by dry and wet deposition of sulfur (68 kg/ha).

B.1.3 Sensitivity

B.1.3.1 Higher plants--

B.1.3.1.1 Symptoms. The damaging effects of sulfur compounds on plants are often accompanied by observable symptoms. The acute symptoms of SO_2 exposure have been documented for a number of plant types (Taylor 1973; Barrett and Benedict 1970; Linzon 1965), as have the acute effects of H_2S exposure (Thompson and Kats 1978; Benedict and Breen 1955). For example, acute SO_2 exposure of broadleaf plants may cause partial necrosis and discoloration in specific parts of the leaf, possibly leading to shedding of the leaf. Less is known of the acute effects of exposure to H_2SO_4 mist or aerosol, but some symptoms have been defined (Middleton et al. 1950, 1958, 1965; Lang and Krupa 1977, 1978).

Chronic exposure of broadleaf plants to low concentrations of SO_2 may lead to varied color patterns, suggesting senescence. As with acute exposure, the symptomatic effects of which may occur simultaneously, leaf shedding may result (U.S. Environmental Protection Agency 1973). There is little information on the symptomatic effects of chronic exposure to H_2S or H_2SO_4 mists and aerosols.

Certain physiological disturbances unaccompanied by apparent signs of exposure may occur. For example, Keller (1977) found that SO_2 reduced photosynthesis in fir and spruce that showed no visible symptoms.

B.1.3.1.2 Dose response. Information on SO_2 doses causing acute injury to sensitive plant species is summarized in Table B-2. Table B-3 provides some of the data on growth reductions in vegetation exposed to sulfur dioxide for several hours. Figure B-2 depicts an effects model of threshold injury from SO_2 exposure.

Table B-4 shows some of the results of studies on average SO_2 concentrations inducing a reduction in epiphytic lichens and bryophytes. Lichens are well accepted as bioindicators of sulfur dioxide pollution of the atmosphere.

Only recently has some information been found regarding dose-response relationships for hydrogen sulfide (Thompson and Kats 1978). Tables B-5 through B-8 summarize the effects of prolonged H_2S exposures on vegetation (Thompson and Kats 1978).

At this time, no definitive information is available on dose-response relationships for submicron sulfuric acid aerosols and acute injury on vegetation.

Table B-2. SULFUR DIOXIDE CONCENTRATIONS CAUSING THRESHOLD INJURY TO VARIOUS SENSITIVITY GROUPINGS OF VEGETATION[a]

Maximum average concentration	Sensitivity grouping Sensitive ($ppm\ SO_2$)	 Intermediate ($ppm\ SO_2$)	 Resistant ($ppm\ SO_2$)
Peak	1.0-1.5	1.5-2.0	>2.0
1-Hr	0.5-1.0	1.0-2.0	>2.0
3-Hr	0.3-0.6	0.6-0.8	>0.8
	Ragweeds	Maples	White oaks
	Legumes	Locust	Potato
	Blackberry	Sweetgum	Upland cotton
	Southern pines	Cherry	Corn
	Red and black oaks	Elms	Dogwood
	White ash	Tuliptree	Peach
	Sumacs	Many crop and garden species	

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[a] Based on observations over a 20-year period of visible injury occurring on over 120 species growing in the vicinities of coal-fired power plants in the southeastern United States.

Source: Jones et al. (1974).

Table B-3. GROWTH REDUCTION IN VEGETATION EXPOSED TO SULFUR DIOXIDE IN FIELD EXPOSURE CHAMBERS FOR SHORT TIME PERIODS

	Concentration[a]			
Species	μg/m³	ppm	Exposure time (hr)	Effect
Crimson clover (*Tribolium incarnatum* L.)	2489	0.95	8	Reduced growth
Red clover (*Trifolium pratense* L.)	2489	0.95	12	Reduced growth
Italian rye (*Lolium multiflorum* Lmk.)	2489	0.95	12	Reduced growth
Mixtures of:				
T. pratense and	2489	0.95	12	Reduced growth
L. multiflorum				Growth not affected
Vetch (*Vicia sativa* L. and *V. faba* L.), pea (*Pisum arvense* L.), and lupine (*Lupinus lentens* L.)	996	0.38	48	Reduced growth for all species

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[a] Average concentrations over the reported time periods. Inaccuracies associated with instrumentation result in deviations as great as ±10 percent.

Source: U.S. Environmental Protection Agency (1973).

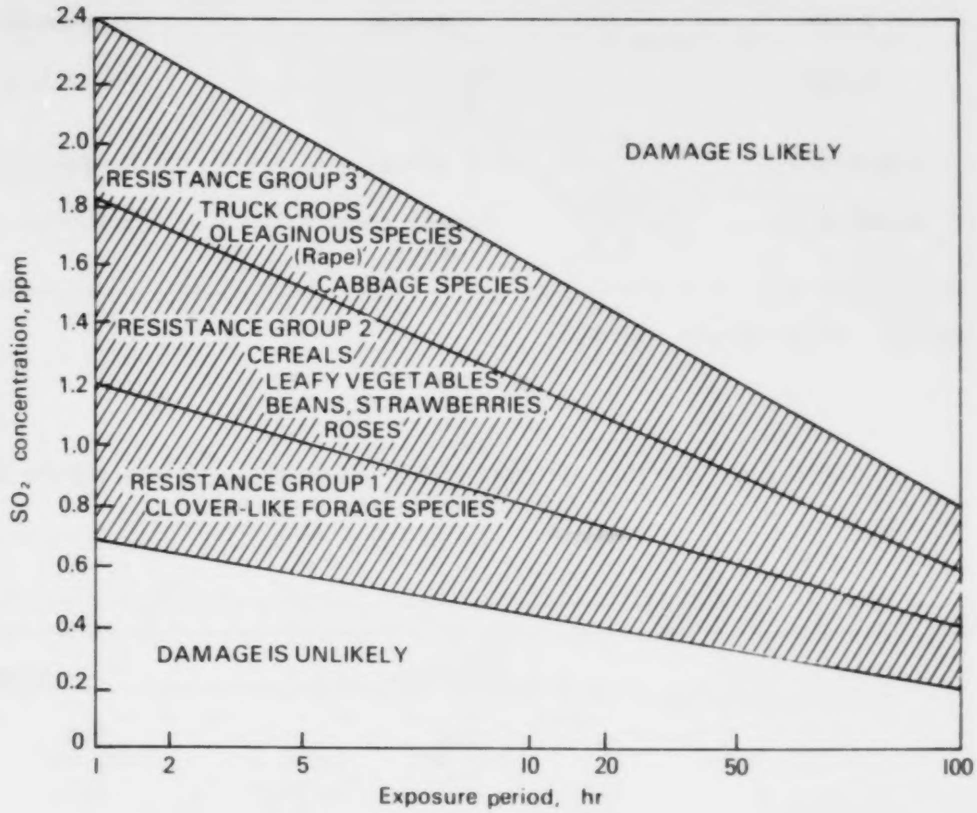

Figure B-2. Sulfur dioxide concentrations that may produce threshold injury (hatched area) to vegetation with continuous exposure for various periods of time.

Source: Zahn (1961).

Table B-4. AVERAGE SULFUR DIOXIDE CONCENTRATIONS RESULTING IN A DECREASE IN THE NUMBER OF EPIPHYTIC LICHEN AND BRYOPHYTE SPECIES

SO_2 concentration, ppm	Averaging period	Research area
0.015	Annual	Stockholm, Sweden
0.020	Winter (Oct.-April)	Newcastle, England
0.015-0.03	Annual	Belfast, Ireland
0.005-0.01	Summer (May-Oct.)	Sudbury, Canada

Source: Jones et al. (1974).

Table B-5. EFFECTS OF CONTINUOUS H_2S FUMIGATION[a] ON GROWTH AND SULFUR ACCUMULATION IN ALFALFA

		Eldorado		Hayden	
Cutting	H_2S, ppb	Avg dry wt/ pot, g	Total S as $SO_4^{=}$, %	Avg dry wt/ pot, g	Total S as $SO_4^{=}$, %
#1	0	52 y[b]	1.03	52 x	0.92
	30	51 y	1.23	52 x	1.36
	300	42 y	2.45	32 y	2.44
	3000	16 z	4.85	11 z	5.20
#2	0	45 y	0.94	46 y	1.00
	30	46 y	1.10	43 y	1.29
	300	31 z	3.00	28 z	3.41

[a] Days of fumigation: 28 to 35.
[b] Values followed by different letters are different at the 1% level.

Source: Thompson and Kats (1978).

Table B-6. EFFECTS OF INCREASING LEVELS OF H_2S ON CANE LENGTH, DEAD LENGTH, AND DRY WEIGHT OF THOMPSON SEEDLESS GRAPES[a]

H_2S, ppb	Total length, cm	Dead length, cm	% Dead	Total dry wt, g	Total sulfur as SO_4 in leaves, %
0	1252.9 y[b]	572.1 x	63.1 y	145.0 x	0.78
30	1160.0 y	422.2 x	53.3 y	143.1 x	1.26
300	1090.1 y	212.4 x	18.7 z	72.5 y	3.33
3000	673.5 z	191.3 z	15.5 z	38.0 z	4.50

[a] Days of fumigation: 117.
[b] Values followed by different letters are different at the 1% level.

Source: Thompson and Kats (1978).

Table B-7. EFFECTS OF INCREASING LEVELS OF H_2S ON LEAF AND CANE WEIGHTS OF GRAPES[a]

H_2S, ppb	Leaves		Canes	
	Fresh wt, g	Dry wt, g	Fresh wt, g	Dry wt, g
0	265.6 a[b]	78.6 a	267.2 a	123.8 a
30	375.6 b	97.0 a	251.8 a	115.8 ab
100	298.4 b	80.6 a	219.6 a	86.0 bc
300	260.4 a	57.0 b	152.0 b	62.8 c

[a] Days of fumigation: 145.
[b] Values followed by different letters are different at the 5% level.

Source: Thompson and Kats (1978).

Table B-8. YIELD OF LEAVES, ROOTS, AND SUGAR CONTENT OF SUGAR BEETS EXPOSED TO INCREASING LEVELS OF H_2S[a]

H_2S, ppb	Leaves Fresh wt, g	Leaves Dry wt, g	Leaves Sulfate, %
0	149.6 bc	19.8 bc	0.66
30	210.5 a	25.2 b	0.93
100	200.8 ab	23.3 b	1.33
300	127.6 c	15.2 c	1.88
	Roots Fresh wt, g	Sugar, %	Sulfate, %
0	291.3 bc	19.3 a	0.05
30	440.6 a	18.3 a	0.06
100	370.7 ab	18.2 a	0.08
300	219.6 c	15.8 b	0.12
	Total, 3 beets/pot Fresh wt, leaves, g	Dry wt, leaves, g	Fresh wt, roots, g
0	1242.0 c	126.2 c	643.0 bc
30	2037.6 a	194.0 a	881.0 ab
100	1874.8	176.8 ab	1034.2 a
300	1640.8 abc	137.0 bc	502.2 c

[a] Days of fumigation: 123.
[b] Values followed by different letters are different at the 5% level.

Source: Thompson and Kats (1978).

Shriner and McLaughlin (personal communication, 1977) evaluated the effects of long-term exposure of beans to COS (carbonyl sulfide). Root biomass ranged from 0-21 percent of the controls when the plants were exposed to 0 and 0.30 ppm COS, respectively, for 3 hr per day, 4 days per week for 4 weeks (total, 48 hr). According to the investigators, this dose appeared to be slightly below that required for consistent reduction in growth or yield of bean.

B.1.3.1.3 *Sensitive species*. For a variety of reasons, certain plants have become well known as bioindicators for the detection of air pollution. Their low injury thresholds, specificity of response to certain pollutants, wide geographic range, low cost, ease of operation, and availability for use throughout all study areas have been suggested as reasons for their use over more costly and sophisticate instrumentation. Plant bioindicators will not replace such instrumentation for monitoring purposes, but such plants have been used to assist in evaluating pollutan levels present in a given area. Table B-9 presents a partial listing of sensitive plants identified as potential bioindicators.

Extensive efforts have also been made to develop certain plant species as bioindicators. Perhaps the most widely examined plants for this use are eastern white pine (*Pinus strobus*) and numerous species of lichens. Table B-10 shows a listing of plants most commonly developed for use as bioindicators.

B.1.3.2 *Lower plants* - *microorganisms*--In recent years, various studies have been conducted to examine the effects of SO_2 on microorganisms, including fungi, algae, and bacteria. Under laboratory conditions, high levels of exposure have produced marked effects, such as reduced growth. Generally, the effects of SO_2 at ambient

Table B-9. PARTIAL LIST OF PLANTS KNOWN TO BE SENSITIVE TO SO_2 UNDER FIELD EXPOSURE CONDITIONS

Cultivated crops

Alfalfa - *Medicago sativa* L.
Barley - *Hordeum vulgare* L.
Bean - *Phaseolus vulgaris* L.
Bluegrass - *Poa annua* L.
Cabbage - *Brassica oleracea* L.
Cucumber - *Cucumis salivus* L.
Oats - *Avena sativa* L.
Pea - *Pisum sativum* L.
Radish - *Raphanus sativus* L.
Rhubarb - *Rheum rhaporiticum* L.
Rye - *Secale cereale* L.
Soybean - *Glycine max* Merr.
Spinach - *Spinacia oleracea* L.
Tobacco - *Nicotiana tobacum* L.

Weed species

Big-leaved aster - *Aster macrophyllum*
Binweed - *Convolvulus arvensis*
Blueberry - *Vaccinium angustifolium*
Curly dock - *Rumex crispus*
Dandelion - *Taraxacum officianle*
Fleabane - *Erigeron canadensis*
Lambs quarter - *Chenopodium album*
Pigweed - *Amaranthus retroflexus*
Plantain - *Plantago major*
Ragweed - *Ambrosia* spp.
Raspberry - *Rhubus* spp.
Smartweed - *Polygorium* spp.
Wildgrape - *Vitis* spp.

Forest trees

Ash, mountain - *Sorbus aucuparia*
Ash, white - *Fraxinus americana*
Aspen, large tooth - *Populus grandidentata*
Birch - *Betula* spp.
Elm - *Ulmus* spp.
Fir, Douglas - *Pseudotsugae menziesii*
Hybrid poplar - *Populus*
Larch - *Larix* spp.
Maple - *Acer* spp.
Pine, jack - *Pinus ponderosae*
Pine, ponderosa - *Pinus banksiana*
Pine, eastern white - *Pinus strobus*
Willow - *Salix* spp.

Ornamentals and flowers

Aster - *Aster bigelovii*
Azalea - *Rhododendron* spp.
Bachelor's button - *Centarea cyanus*
Begonia - *Begonia* spp.
Cosmos - *Cosmos bipinnatus*
Four o'clock - *Mirabilis jalapa*
Morningglory - *Ipomoea purpurea*
Petunia - *Petunia hybrida*
Sweet pea - *Lathyrus odoratus*
Verbena - *Verbena canadensis*
Violet - *Viola* spp.
Zinnia - *Zinnea elegans*

Table B-10. LISTING OF PLANTS THAT HAVE BEEN SELECTED FOR SPECIFIC USE AS BIOINDICATORS OF SO_2 IN VARIOUS INVESTIGATIONS UNDER FIELD CONDITIONS

Plant	Latin name
Alfalfa	*Medicago sativum*
Ash, white	*Fraxinus americanum*
Beans	*Phaseolus vulgaris*
Hybrid poplar	*Populus deltoides* cv. *Angulata* x *Trichocarpa*
Petunia (white flowering)	*Petunia hybrida*
Pine, eastern white	*Pinus strobus*
Pine, loblolly	*Pinus taeda*
Sycamore	*Platanus occidentalis*
Tobacco	*Nicotiana tobacum*

levels appear minimal. However, the overall effect on the numerous plant/environment/microorganism interactions must not be overlooked. Since SO_2 affects soil pH, leaf surfaces, and related factors, the associated microorganisms may also be affected. For example, Mrvka and Grunda (1969) found that bacterial populations were drastically reduced where soil pH, soil potassium, and soil magnesium were likewise decreased.

B.1.3.3 Environmental factors--Plants are subjected to many changes in the environment during their life cycle. Thus, it is important to understand the response of plants to sulfur compounds under varying environmental conditions. Parameters of interest consist of temperature, light, humidity, carbon dioxide concentration, soil moisture, and soil nutrients. Almost all the published literature on this subject pertains to SO_2.

Temperature before, during, and after exposure generally appears to be directly related to the SO_2 sensitivity of trees (Davis 1975). The European literature generally considers SO_2 sensitivity to be directly related to temperatures above 40°F (4°C) (Van Haut and Stratmann 1970). More recently, Heck and Dunning (1978) showed increased sensitivity of oaks to SO_2 at higher growth temperatures within a range of 18-30°C.

Plant sensitivity tends to increase with increasing humidity. Oaks are more sensitive to SO_2 at 80 percent than at 55 percent relative humidity (Heck and Dunning 1978).

Zimmerman (1955) reported that SO_2 sensitivity of plants was directly related to increasing light intensity, up to about 3000 ft-c. Plants grown in direct sunlight prior to exposure were more resistant than plants grown under a 65 percent reductio in intensity. Similarly, minimum plant injury occurs in the field when bright sunn days follow an SO_2 fumigation (see Davis 1975).

Plants are more sensitive to SO_2 when adequate soil moisture is available for normal plant growth (U.S. Environmental Protection Agency 1973). Plant resistance increases as the soil moisture content approaches the wilting point.

Plant resistance increases with increased soil fertilization in rape, spinach, and radish (U.S. Environmental Protection Agency 1973). In contrast, deficiencies of nitrogen and sulfur were correlated with increased resistance in tobacco and tomato (Leone and Brennan 1972).

Few studies have evaluated the influence of soil structure, temperature, aeration, and related variables on response to SO_2. In studies involving three plant species and four soil types, Guderian (1971) found that plant injury varied depending on soil type, nitrogen applications, and plant species.

B.1.4 Interactive Effects

B.1.4.1 Synergism of sulfur compounds with other pollutants--In the ambient atmosphere, specific air pollutants do not occur individually but in combination with other pollutants. Interactions between these pollutants cause vegetational injury classified as antagonistic (less injury), additive (increased injury), or synergistic (greater than additive injury). Most of the research on pollutant combinations has focused on SO_2 and O_3 (ozone), with less emphasis on SO_2 and NO_2 (nitrogen dioxide), SO_2 and F (fluorides), and SO_2-NO_2-O_3 combinations.

Menser and Heggestad (1966) were the first to report on O_3-SO_2 synergistic effects on tobacco plants. Later Tingey et al. (1973) used ambient levels of SO_2 and O_3 individually and in combination on 11 different crop species and demonstrated several synergistic responses. Most of the work in this area has focused on foliar symptoms, although Tingey and Reinert (1975) showed additive effects and greater reductions after low exposures of SO_2-O_3. Other species that have been studied for sensitivity to SO_2 combinations include the eastern white pine (Gerhold 1977) and several hardwood species (Karnosky 1976).

The studies of SO_2-NO_2 combinations show interactive effects considerably less pronounced than those involving SO_2-O_3 (Reinert 1971; Tingey et al. 1971; Bennett and Hill 1975; Skelly et al. 1972).

SO_2-NO_2-O_3 combinations have been investigated by Kress and Skelly (1977) and by Kress (1978). Significant growth reductions of sycamore and loblolly pine resulted from 6-hr daily doses of 5 pphm O_3, 14 pphm SO_2, and 10 pphm NO_2 over 28 days. In 1973, Fujiwara et al. reported that the addition of NO_2 to a SO_2-O_3 combination did not significantly increase effects already exerted by SO_2-O_3 on peas and spinach.

B.1.4.2 Interaction between sulfur dioxide and plant parasites--Plant disease is caused by the interaction of a plant and a pathogen acting under suitable environmental conditions. Although, in the strict sense, a pollutant can be classified as a pathogen, this section will limit the definition to biological parasites affecting plants. The parasites range from fungi to insects.

In general, sulfur dioxide is harmful to plant parasites, depending on concentration and exposure. In 1973, Heagle reviewed pollutant-parasite research and

Table B-11. EFFECTS OF SULFUR DIOXIDE ON PLANT DISEASES

Pollutant and disease affected	Effects	Pollutant dose	Location
Wheat stem rust	Decreased incidence	Ambient	Sweden
Tree rusts	Decreased incidence	Ambient	Canada
Wood rots	Decreased incidence	Ambient	Czechoslovakia
Needle cast on juniper	Decreased incidence	Ambient	England
Needle cast on larch and pine	Decreased incidence	Ambient	Canada
Rhytisma on maple	Decreased incidence	Ambient	England
Hysterium on alder and birch	Decreased incidence	Ambient	Sweden
Apple scab	Decreased incidence	Ambient	Poland
Oak powdery mildew	Decreased incidence	Ambient	Austria
Lilac powdery mildew	Decreased incidence	Ambient	U.S.A
Rose black spot	Decreased incidence	Ambient	England
Rose black spot	Smaller lesions	0.04 ppm, 48 hr	England
Dwarf mistletoe on larch and pine	Decreased incidence	Ambient	Canada
Armillaria in trees	Increased incidence	Ambient	Canada, Czechoslovakia, Poland, Germany
Wood rots	Increased incidence	Ambient	Czechoslovakia
Needle cast on spruce	Increased incidence	Ambient	Poland

noted certain effects of pollutants on plant parasites. As seen in Table B-11, ambient levels of SO_2 generally decreased the incidence of fungal disease on various forest trees.

Similarly, Hibben and Taylor (1975) observed that mildew infection was reduced by acute and chronic doses of SO_2, which they labeled as fungicidal to that specific mildew. Several other studies also support this fungicidal action in various species (Koch 1935; Majernik 1971; Saunders 1966; Scheffer and Hedgcock 1955; Linzon 1958). In addition, SO_2 is used extensively to prevent grape mold and rot during storage and shipment (Nelson 1958; Nelson and Ahmedullah 1972; Soto et al. 1973).

However, SO_2 does not harm all fungi. Weinstein et al. (1975) showed that while SO_2 decreased the severity of bean rust, it had no effect on tomato blight. Far more important, however, is that SO_2 generally weakens plants, and in doing so makes the plant more vulnerable to parasitic attack (Donaubauer 1968; Jancarik 1961; Kudela and Novakova 1962).

This same selective parasiticidal action holds true for higher organisms. Stewart et al. (1973) reported that SO_2 may or may not repel lice, depending on plant species infested. Weber et al. (1974) reported a similar finding using SO_2 and O_3 singly and in combination against nematode infestation.

Although no direct effects of SO_2 on plant pathogenic bacteria have been reported, certain indirect effects may be exerted by changes in soil acidity and nutrient change (Heagle 1973). Shriner (1978) reported on the effects of simulated acid rain on host-parasite interactions in plant disease development.

B.1.5 Physiological Effects

B.1.5.1 Ingress of SO_2 into plants and stomatal influences--The stomates of leaves are the major avenue of SO_2 entrance into plants (Guderian 1977; Katz 1949 Thomas 1951; Thomas and Hendricks 1956; U.S. Environmental Protection Agency 1973 but there is no general agreement regarding the role of stomata in plant sensitiv There appear to be numerous factors which govern the mechanism of stomatal openin and closing. Ting and Dugger (1968) have reported that there is no linear relati ship between pore size and permeability of leaves to gases. Gas exchange through stomata is a dynamic process influenced by gas diffusion coefficient, pressure in and outside the leaf, stomatal resistance, and other variables. In addition, physical factors such as light, leaf surface moisture, relative humidity, and soil moisture may actually play the major role in plant sensitivity, while SO_2 enters leaf by passive means (Setterstrom and Zimmerman 1939; Zimmerman 1950; Meidner an Mansfield 1968; Spedding 1969; Domes 1971). SO_2 may also enter plants through the leaf cuticle during periods of stomatal closure in the presence of dew (Garland an Branson 1978; Fowler 1976).

The numbers and size of stomates of the individual species or species genotyp could influence pollutant ingress. For example, Nikolaevskii (1971) reported that SO_2 damage to several woody plants was greatest at midday even though the degree o stomatal opening did not change during the diurnal cycle. He concluded that certain sensitive species generally had a larger number of stomata/mm^2 of leaf area a a greater degree of natural stomatal opening than did certain resistant trees.

Ricks and Williams (1974) looked at the leaves of oak (*Quercus petraea*) in an area of woodland subjected to a differential gradient of industrial particulate pollution and compared them to leaves from an unpolluted rural woodland. They found that an accumulation of particles on the lower surfaces of the leaves mechanically prevented normal stomatal closure at night. Overall rates of water loss were increased as diffusive resistance decreased, and the uptake of SO_2 was subsequently enhanced for extended periods of time.

B.1.5.2 Stomatal response to SO_2--Jensen and Koslowski (1975) found that sulfur absorption rates varied among several species of forest trees fumigated with SO_2, as did the onset of resistance to absorption. The sulfur content of the foliage of all species increased, and at 8 days following fumigation with radioactively labeled SO_2, varying amounts were found throughout the plants, including the roots. It was observed that a plant's tolerance to SO_2 may depend less on the amount of pollutant absorbed than on the ability of the plant to move SO_2 out of the leaf and into other plant tissues.

While SO_2-induced closure has been observed elsewhere (Bonte et al. 1975), other reports indicate that SO_2 may exert an opposite effect under certain conditions. For example, stomatal opening or closing in response to SO_2 may depend upon the duration of exposure (Buron and Cornic 1973), relative humidity (Majernik and Mansfield 1970; Mansfield and Majernik 1970), or drought versus nondrought conditions (Schramel 1975). Unsworth et al. (1972) reported that the stomatal opening of the leaves of many crops was increased by ambient SO_2 levels. Uniformly increasing the SO_2 concentration up to 2.0 ppm on beans and corn reduced stomatal diffusive resistance, with young leaves exhibiting the lowest resistances. The effect of SO_2 on stomatal resistance was greater on all leaves of water-stressed plants.

For further discussion on variables affecting plant sensitivity to SO_2, see "Environmental factors" (Section B.1.3.3 of this appendix).

B.1.6 Acidic Rain

B.1.6.1 Formation of acidic rain--The acidity of rainfall in many geographic areas has been increasing in recent years. This increase appears to stem from the atmospheric loading of SO_x and NO_x, as emitted from fossil fuel combustion. Although a rain pH of 2.1 was noted by Likens and Bormann (1974), the mean pH for precipitation nationwide ranges from 4.0 in the New England States to 6.1 in the Pacific Southwest (see Figure B-3). For comparison, the pH of an unpolluted atmosphere would be 5.7.

Sulfur was first recognized as a component of rainwater by Wilson in 1926. Since then, over 22 different species of atmospheric sulfur have been suggested. Of these, SO_2, $SO_4^{=}$, $SO_3^{=}$, and S_2O_6 can contribute to the acidity of rain. Unfortunately, despite years of research, the chemistry of sulfur in the atmosphere, particularly sulfate formation, is only partially understood. Nevertheless some of the forms of sulfur found in rain have been characterized. Hales (1977) found that up to 30 percent of the sulfur found in rain occurred as sulfite. Essman and Fergus (1975) suggested that the major sulfur source of acidic rain in Pennsylvania occurred as dithionate (S_2O_6). Generally, the data seem to indicate that the acidity of rain and the role each sulfur species plays may vary with time and place.

Gambell and Fisher (1966) argue that anthropogenic sulfur is not the controlling factor of rain acidity. However, Leland (1952) and others note an increase in sulfate during the winter months, which they attribute to increased coal and oil burning. This increase could contribute to rain acidity. Many researchers believe that sulfate is formed through a catalytic oxidation process (Georgii 1970; Junge 1960; Meszaros 1970; Rodhe 1970). One probable method of formation is the "SO_2-NH_3-H_2O" system.

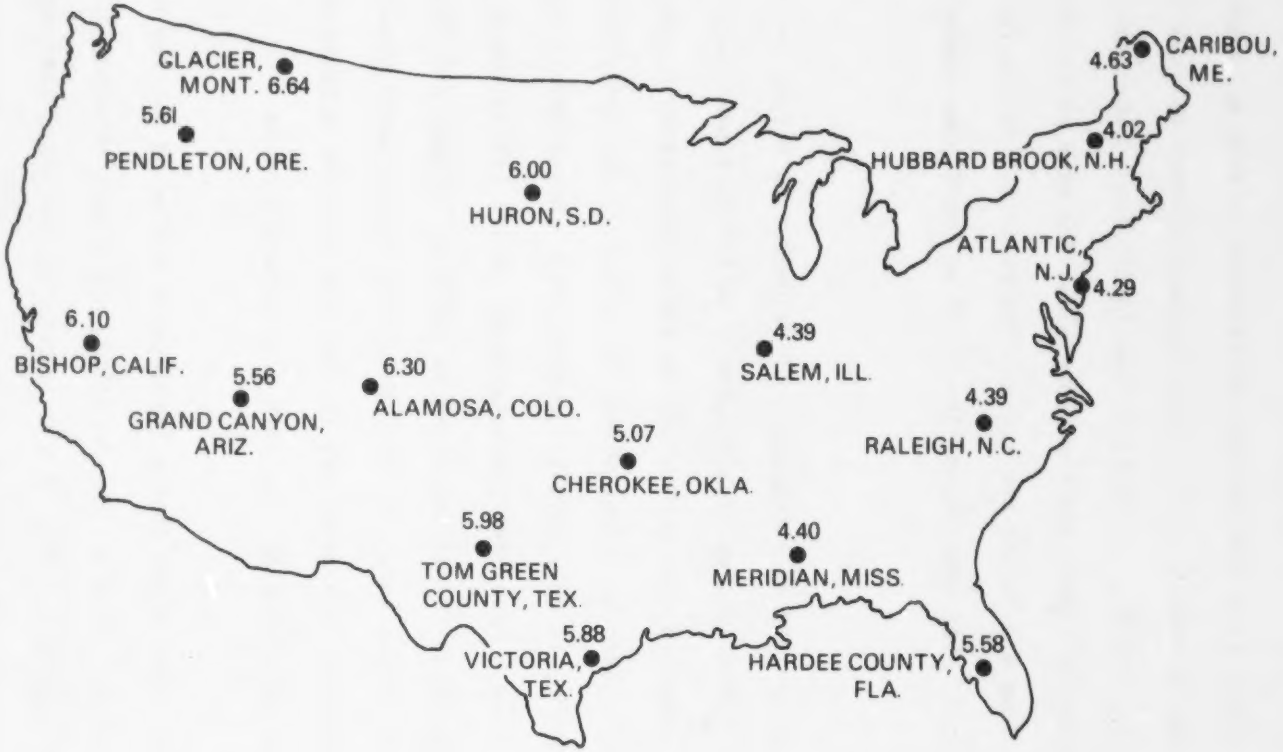
CARIBOU, ME. 4.63
HUBBARD BROOK, N.H. 4.02
ATLANTIC, N.J. 4.29
GLACIER, MONT. 6.64
PENDLETON, ORE. 5.6I
HURON, S.D. 6.00
BISHOP, CALIF. 6.10
GRAND CANYON, ARIZ. 5.56
ALAMOSA, COLO. 6.30
SALEM, ILL. 4.39
RALEIGH, N.C. 4.39
CHEROKEE, OKLA. 5.07
TOM GREEN COUNTY, TEX. 5.98
MERIDIAN, MISS. 4.40
VICTORIA, TEX. 5.88
HARDEE COUNTY, FLA. 5.58

Figure B-3. Weighted mean pH of precipitation over the United States for 1972-3.

Source: J. Miller, personal communication

Several Scandinavian investigators believe that most of the sulfur found in precipitation arises from global, upper-atmospheric sources rather than anthropog sources (Oden 1964). In support of this belief, Gambell and Fisher (1964) found the concentration of sulfate in the lower atmosphere remained at high levels thro out the course of rainstorms.

However, Bielke and Georgii (1968) estimate that the concentration of SO_2 in the lowest kilometer of the atmosphere exceeds that in higher altitudes according the following table:

	SO_2	Aerosol $SO_4^=$
Rainout (cloud contribution)	5%	20%
Washout (sub-cloud contribution)	70%	5%

The relatively high SO_2 levels in the lower atmosphere could contribute extensivel to the gaseous sulfate in rain. But Miller and de Pena (1972), who measured the contribution of scavenged gas to the sulfate content of rain, concluded that washe out particles are the major contributors.

Most of the acid rain studies have been based on correlations between pH and sulfate deposition (Galloway and Parker, unpublished). Johnson et al. (1972) and Krupa (1976) presented data indicating that atmospheric sulfuric acid concentratio are not sufficient to reduce rain pH to below 3.0. Johnson et al. (1972) calculat that 3-4 mg/liter sulfate would lower the pH of rain to 4.4-4.1. Consequently, other active anions must be present to account for the low observed pH of rain.

Likens et al. (1976) suggest that anthropogenic $SO_4^{=}$ and NO_3^{-} (as sulfuric acid and nitric acid) contribute most to the acidity of rain.

Studies at the University of Maryland have measured rain pH and composition in the Washington, D.C., metropolitan area at various locations, including areas near power plant stacks. One particular study by Muhlbaier (1978) involved measuring rain pH at and near a power plant fueled by coal and oil. Sufficient quantities of NO_x and SO_x were found in the rainwater to account for the low rain pH. In addition, since $SO_4^{=}$ dominated atmospheric acid species, Muhlbaier concluded that in the microclimate around the power plant, sulfate concentrations correlated well with the acidity of the rain.

Although Frohliger (1976) found that the pK values indicate that rain has a weak acid character, Krupa et al. (1976) showed that rain contained both strong, volatile acids and weak, nonvolatile acids. Nevertheless, measurements made by Oden (1968), Anderson (1969), and Krupa et al. (1976) could not correlate change in pH with sulfate concentrations in the atmosphere. An important fact in understanding acid rain components was brought up by Whitby (1977), who noted that the non-neutralized sulfate (i.e., sulfuric acid) particles (0.01-0.1 μm in diameter) are too small to be removed and deposited by rain.

Although mobile sources produce significant amounts of nitrogen oxides which could increase rain acidity, the sulfate particles they produce, particularly sulfuric acid, not only are insignificant in amount but also are in the submicron size range.

B.1.6.2 Vegetational effects of acidic rain--Acidic rain has been implicated in the increased leaching of nutrients from soils (Overrein 1972) and decreased pH

Table B-12. EFFECTS OF ARTIFICIAL ACID RAIN ON VARIOUS PLANT SPECIES

Researchers	Plant species	Effects
Gordon (1972)	Pine needles	50% growth reduction
Shriner (1974)	Oak and bean leaves	Cuticle erosion
Wood and Bormann (1975)	Yellow birch and sugar maple seedlings	Foliar spot necrosis; irregular leaf development
Wood and Pennypaker (unpublished)	Scotch pine	Needle necrosis
Evans (personal communication, 1978)	Bean plants	Leaf lesions; decreased leaf expansion; premature defoliation; fewer and smaller pods/plant
Shriner (personal communication, 1978)	Bean plants	No change in foliage dry wt In combination with ozone - reduction in foliage dry wt

values of lakes and streams (Oden and Ahl 1970). Little is known, however, of the direct effects of acidic precipitation on terrestrial vegetation. A review of the literature reveals no published reports demonstrating vegetational injury from naturally occurring acidic rainfall. Several investigators, however, have reported direct injury of various plant species by artificially applied acid mists and acidic solutions (Ferenbaugh 1976; Shriner et al. 1974; Wood and Bormann 1974) (see Table B-12). According to Wood (1975), the changes acidic rain may induce in natural ecosystems are likely to be widespread but difficult to detect over short periods of time.

The available literature indicates that pollution in rainfall may cause direct injury to foliage, deposition of other harmful substances on the plant tissue, effects on host-parasite relationships, and effects on the soil.

Crowther and Ruston (1911) were the earliest investigators to ascribe plant damage to the acidic nature of polluted precipitation in Leeds, England. They described direct leaf toxicity and "less nutritious timothy grass" as manifestations of the pollution in rain. Syratt and Wainstall (1968) suggested that the acidic nature of SO_2 at high humidity (sulfurous acid) degrades chlorophyll. Oden (1963) stated that the acidic nature of most sulfurous compounds may plasmolyze plant cells at high concentrations and destroy chlorophyll at lower concentrations. He also noted that acidic rains significantly affect coniferous vegetation because of the accumulative properties of the needles. Likens et al. (1972) reported that Sweden suffers an annual reduction in forest growth as a result of acidic rainfall.

Shriner (personal communication, 1978) studied the effects of simulated acidic rain (sulfuric acid) on host-parasite interactions at pH 3.2. Acid treatments

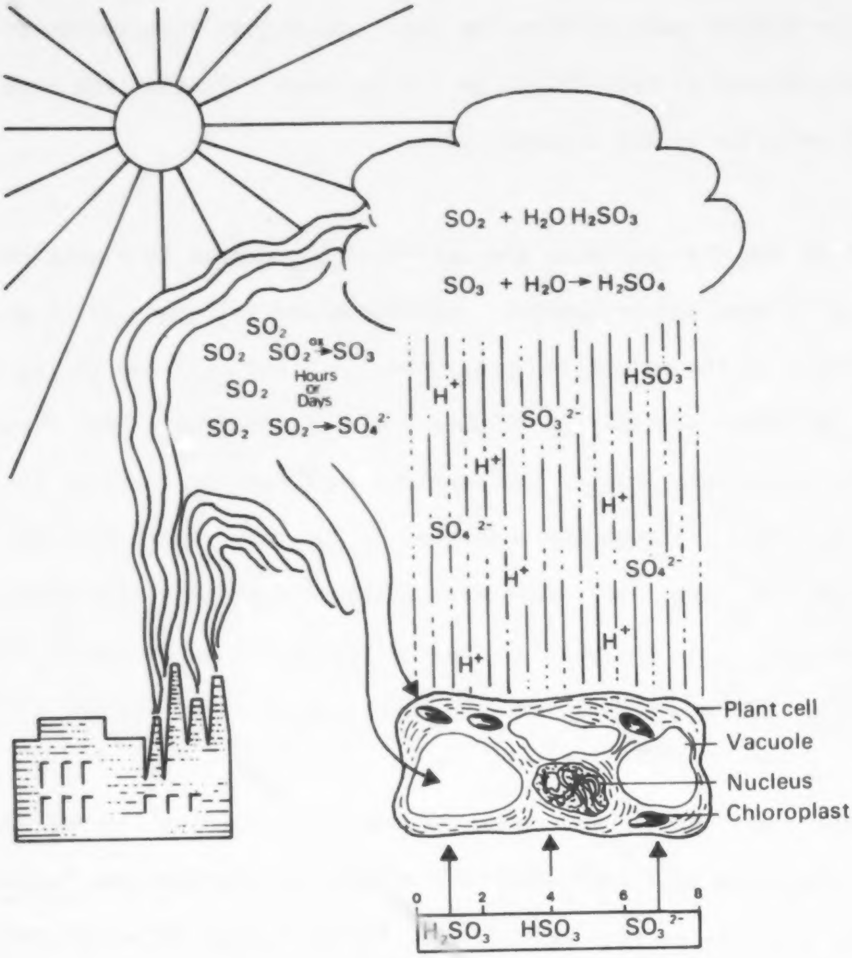
$SO_2 + H_2O\ H_2SO_3$
$SO_3 + H_2O \rightarrow H_2SO_4$
SO_2
SO_2 $SO_2 \xrightarrow{or} SO_3$
SO_2 Hours or Days
SO_2 $SO_2 \rightarrow SO_4^{2-}$
H^+
HSO_3^-
SO_3^{2-}
SO_4^{2-}
Plant cell
Vacuole
Nucleus
Chloroplast
0 2 4 6 8
H_2SO_3 HSO_3 SO_3^{2-}

Figure B-4. Emissions and transformation of SO_2 and SO_3. The figure shows the probable transformations of SO_2 in air and water. The arrows into the cell give an approximate picture of the permeability of the various sulfurous compounds. The distribution of the various dissociation states of H_2SO_3 at differen pH values is shown in the pH scale below the cell.

Source: Sundstrom and Hallgren (1973).

resulted in: an 86 percent inhibition in the number of telia produced by the oak-pine rust on willow oak; a 66 percent inhibition in the reproduction of a root nematode on field-grown kidney beans; a 29 percent decrease in the leaf area of field-grown kidney beans affected by rust; and either stimulation or inhibition in the development of halo blight on kidney beans. In the last case, the effect varied in relation to the disease cycle.

Much of the discussion on the action of SO_2 damage to plants has arisen from studies on lichens and bryophytes. Sundstrom and Hallgren (1973) portray the "probable" picture of the relationship between the plant cell and the sulfur pollution relative to lichen sensitivity (Figure B-4). Accordingly, they state: "It is reasonable to suppose that in the event of sulfurous pollutions, the transport of SO_2 or H_2SO_3 and HSO_3^- through the membranes is much more rapid than the uptake of the more charged $SO_3^=$ ion. This selective uptake of undissociated molecules, and to some degree HSO_3^-, is followed by their dissociation in the cell, resulting in an accumulation of charged HSO_3^- and $SO_3^=$ ions and an acidification of the cell."

Mounting acid inputs may cause decreases in the fertility of forest soils. It has been suggested that increased rain acidity accelerates the leaching of potassium, magnesium, and calcium (Overrein 1972). Field studies in Sweden correlate increased acidic rain inputs with decreases in soil pH (Oden et al. 1972).

Conversely, increased soil fertility may also result from acidic rain. For example, this rain may add nitrate and sulfate ions, common components of chemical fertilizers. In fact, Wood and Bormann (cf. Wood 1975) found that mean productivity of white pine seedlings subjected to artificial rains containing sulfuric, nitric, and hydrochloric acids was 20 percent greater at pH 2.3 than at less acidic concentrations.

Plants constitute rather effective filters, according to Eaton et al. (1973). They observed that during growing seasons, forest canopies filter 90 percent of the hydrogen ions from pH 4.0 rainwater. As a result, less acidic solutions (pH 5.0) reach the forest floor.

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B.2 EFFECT ON NONBIOLOGICAL MATERIALS

B.2.1 Introduction

The damaging effects of air pollution on nonliving objects can be quantified in economic terms by a materials damage estimate. This estimate, based on complex methods of data selection and analysis, helps to assess the relative costs and benefits of attempts to reduce or prevent pollution. On the basis of studies conducted to date, a best-guess estimate of materials damage from sulfur-bearing compounds emitted by mobile sources (2 percent contribution) is $30 million annually. It should be noted, however, that the state of the art in making materials damage estimates is relativity undeveloped. The accuracy and validity of these estimates could be improved greatly by refining the economic damage function, which is a measure of the cost of damage relative to the degree of pollution. Better methods for assessing various indirect costs are needed as well.

Mobile sources account nationwide for an average of less than 2 percent of all manmade sulfur compound emissions (stationary sources account for most sulfur emissions). There are no data relating materials damage to mobile sources per se, only a large body of data relating materials damage to specific levels of certain sulfur-bearing compounds. Since it is not possible in ambient air measurements to identify unequivocally the source of these compounds, the only available way to estimate damage by a sulfur-bearing compound from mobile sources is to multiply the overall damage values by the relative proportion of mobile sources to all sources (2 percent). No studies have been conducted to isolate the relative contribution of mobile versus nonmobile sources in particular areas, such as roadways, where the mobile source contribution might be much higher than the nonmobile source contribution.

In terms of potential damage to materials, the most significant sulfur compounds emitted from mobile sources are sulfur dioxide (SO_2), hydrogen sulfide (H_2S), sulfuric acid (H_2SO_4), and absorbed sulfates (SO_4). Less significant species such as organic sulfides, disulfides, and thiophenes are present, if at all, at relatively low concentrations. Since these organic compounds react minimally with common nonliving materials, they are not considered in this report.

It is important to consider damage to materials by the type of sulfur compound emitted by mobile sources. The various species of sulfur compounds emitted directly from these sources are often transformed into other species. These transformed products may have damaging effects different from those of the original compounds. Therefore, knowing what compounds are emitted is not always sufficient to predict subsequent damage to materials.

B.2.2 Physical Damage

B.2.2.1 Types and mechanisms of damage--Generally, air pollutants damage materials by five principal mechanisms: abrasion, direct and indirect chemical attack, corrosion, and particle deposition and removal.

There is insignificant damage from abrasion attributable to mobile source pollution. Direct chemical attack can occur, however, including the etching of metallic surfaces by acid mists (e.g., H_2SO_4), blackening of lead-based paints (e.g., by H_2S), or leaching or spalling of carbonate building stone (e.g., by SO_2). Indirect chemical attack can also occur in certain materials that absorb pollutants which cause damage when they undergo chemical change.

Some atmospheric pollutants speed up the rate of corrosion considerably. One particular corrosion mechanism, an electrochemical process, is responsible for the deterioration of most ferrous metals. When exposed to pollutants in the atmosphere, the metallic surfaces form numerous tiny electrochemical cells. Their reaction with water, even as a molecular layer on a surface that appears dry, provides an impetus force for corrosive action, and the metal dissolves away. Another corrosive mechanism involves removing protective layers from materials surfaces. For example, the green patina on copper exposed to the atmosphere is attacked by H_2S, which eventually causes the surface to flake off and expose the metal beneath to additional damage. Another, more important example is the destruction of galvanized coatings by the acid species.

The most common effect of particle deposition on fabrics, painted surfaces, and building materials is discoloration, occurring directly by accumulation of the particles themselves or indirectly by other pollutants absorbed by the particles. Cleaning discolored materials can be costly and can further damage or shorten the useful life of the object involved.

B.2.2.2 Environmental factors--Damage from pollutants can occur through synergistic interaction with natural factors such as moisture, particles, temperature, sunlight, air movement, physical position, and biological action.

Without moisture, little if any atmospheric corrosion would occur even in highly polluted environments. Ironically, visibly wet surfaces often reduce corrosion rates, probably from the dilution and washing away of the corrosive material. Corrosive agents, however, can accumulate at the lower edge of a metallic object and accelerate corrosion there. For several metals there is a critical atmospheric humidity that determines the corrosion rate in polluted air. It has been determined, for example, that as relative humidity approaches 80 percent, the corrosive effects of SO_2 proceed rapidly.

In moist atmospheres, concentration of salt particles on the surface of metals can greatly accelerate metal corrosion. In addition, heating and cooling can speed up chemical reactions in metal deterioration. For example, at night, metal objects often cool below the temperature of the ambient air and, if the dew point is reached, moisture accumulates, accelerating corrosion. Freezing and thawing in the presence of moisture speeds the deterioration of porous materials such as concrete and building stone. Sunlight can also deteriorate certain materials, such as rubber and painted surfaces. Wind speed is significant in determining whether solid and liquid agents impact on vertical surfaces, settle on horizontal surfaces, or produce abrasion. Air movement can influence the volume of pollutant reaching the surface of a material and, subsequently, the rate or even the type of reaction.

Spatial position is an important variable in atmospheric corrosion. In corrosion tests in which samples were mounted at 30 or 45 degrees from the horizontal, the under surfaces often corroded more rapidly than the upper surfaces. The reason for this difference may have been that upper surfaces were washed by rainfall and were less susceptible to lingering dampness than were under surfaces.

Biological action such as mildew and bacteria can affect materials of natural origin, such as cotton, wood, and leather. Lichens and other plants affect the moisture level of the surface of materials on which they grow.

The order in which substances contact a surface is also significant. For example, if copper is first exposed to ambient air, a thin oxide film develops to help protect it against hydrogen sulfide. But if the copper is exposed to hydrogen sulfide before the protective coat forms, deterioration begins and can proceed to destruction.

B.2.2.3 Physical damage functions--A physical damage function is a mathematical equation relating the damage of a given material to the pollutants and environmental factors that caused the damage: $D = f(P, F, t)$.

The damage (D) is expressed in terms appropriate to the pollutant/material interaction. For example, the corrosion of steel plate would be expressed in units of thickness lost, but the soiling of paint would be stated in units of reflectance. The pollutant level (P) can be expressed in any concentration unit. Since materials damage usually takes some time to develop, average or long-term pollutant levels are often used. The effects of other environmental factors (F) are often included in only a gross way by specifying a type of environment (cold, wet, etc.). However, meteorological parameters can be obtained and used with as much detail as are pollution level data, if desired. Relative humidity, rainfall, time of wetness, sunlight, wind speed and direction, temperature, and other values are available for many locations. Additional environmental factors can be approximated by more detailed geographical descriptions (e.g., New England/coastal/industrial). Time (t) is usually expressed in years. The yearly time scale makes economic comparisons easier and allows more averaging of conditions in changeable climates.

B.2.3 Aesthetic Damage

In many parts of the world, air polluted with sulfur oxide is silently eating away irreplaceable works of art. Some of the finest monuments of antiquity and thousands of pieces of sculpture and carvings on historic buildings such as cathedrals show vivid evidence of the insidious effects of polluted atmospheres. The marble frieze of the Parthenon in Athens, Greece, is a good example: a plaster cast made in 1802 shows relatively minor damage during the first 2240 years, but a photograph of the same marble frieze taken in 1938 is almost unrecognizable because of rapid deterioration during the intervening 136 years of increasing industrialization.

Conservation scientists recognize that a number of environmental agents hasten deterioration. These include weathering (wind, rain, heat, snow, and frost), traffic vibration, molds, lichens, and pigeon droppings. However, sulfur compounds and other air pollutants, the unwanted byproducts of the industrial age, are considered the major villains.

B.2.4 Economic Damage

There are two basic approaches calculating the economic damage attributed to an air pollutant: comparative and analytic (Stankunas et al. 1978). Neither approach is considered completely satisfactory.

The comparative approach studies the lifetime maintenance costs for a given material in use in several ambient environments. For the method to work, the environments should be as similar as possible, except for the pollutant level. The total materials damage costs for each of the locations are used to calculate an economic damage function for the pollutant. However, several factors make the comparative approach difficult to apply. It assumes, for example, that total materials damage costs can be determined without specifying the type of damage. The approach is also highly sensitive to differences in the nonpollutant variables at each location, and it is difficult to find locations that are suitably matched. Environmental and usage factors can differ so much that the whole mechanism of damage changes. Another weakness in the comparative method is the need to examine many environments to cancel random errors. Approximations and simplified assumptions can obscure the results.

The analytic approach depends on laboratory- or field study-derived physical damage functions and ambient air quality information. First, the materials and their specific interactions with the pollutants of interest must be considered. The value of those materials and their use in society must be assessed. The exposure of the materials to pollutants and other environmental factors must be determined, as well as the effects of repair, replacement, protection, and substitution practices. Finally, the appropriate physical damage functions are used to quantify the damage caused by specific pollutants. The costs of that damage relative to the degree of pollution are then calculated, and the total costs attributed to actual pollutant levels are judged. The weakness of the analytic approach is inherent in its dependency on many complex variables, some of which are difficult to assess in quantitative terms.

B.2.4.1 Choice of materials--Thousands of materials are exposed to sulfur emissions from mobile sources. Since a materials damage study cannot account for all damage to all materials, a scheme is needed to identify those materials whose damage has the greatest economic impact. In general, to warrant detailed study a material should both be affected by the pollutant of interest and be economically important.

Annual production figures are of limited usefulness in determining the economic impact of a given material. Such figures do not take into account the use for which the material is intended and do not include the costs associated with that utilization. If only materials with very high annual production were studied, materials with lower total production but high loss rate due to pollutant damage would be missed. This omission would be particularly important if most of the turnover of the lower production material arose from pollutant damage.

B.2.4.2 Value of materials--A material's value to society is difficult to determine, though there are several publications that can be of help. Retail/wholesale values of steel, for example, are noted in the American Metal Market/Metal Working News; paint values are noted in the Kline Guide to the Paint Industry. Actual values of materials are available also through the U.S. Government agencies that contribute to the Annual Consumer Price Index. However, the cost of the materials themselves is only a small part of the costs of pollutant damage.

Determining the cost of material becomes more complex when the cost of labor is included. Most studies simply apply a "labor factor" to the overall cost of the material. Since labor costs of production or installation dominate total costs for most materials, any uncertainties or errors in estimating a labor factor are greatly magnified in subsequent calculations. Also, the labor factor gives no indication of the differences in value that arise from differences in how the materials are used (e.g., assembly line spray painting versus hand painting by a steeplejack).

B.2.4.3 Maintenance--A major cost item that has not been adequately considered in most materials damage studies is maintenance. Some of the early studies completely ignored the fact that maintenance activity is widely performed to mitigate the effects of materials damage. More recent studies have included the concept of maintenance, but only as it relates to loss or degradation of materials. Generally, it can be assumed that if the costs of failure of a given component exceed the cost of preventive measures, these measures will be taken.

In some instances, maintenance may not be cost effective. Uncoated galvanized fencing, for instance, is less expensive to replace than it is to maintain. On the other hand, color-coated galvanized fencing has prepaid maintenance value (as well as aesthetic value). Unless the analytic approach specifically accounts for the principal maintenance-controlling factors, the damage cost estimate it generates is almost useless.

In some cases, protection against air pollution damage may arise from normal maintenance, but it may not be the primary determinant of the maintenance schedule. For example, the protective coating of paint applied to steel bridges, particularly to the roadway surface, will usually be damaged by rock impact, salt, and other such factors long before the paint can be appreciably damaged by air pollution. Similarly, a house may need repainting because of peeling damage caused by moisture. In both cases, maintenance is required because of damage to the paint arising from factors other than air pollution. In essence, then, air pollution is not always the major determinant of the maintenance schedule or cost.

B.2.4.4 Relative lifetimes--The damage by a pollutant must be significant relative to the normal lifetime of the material. Children's shoes are rarely discarded because of SO_2 damage, although the leather may have a measurable damage function. There are hundreds of similar examples. To assess damage costs, it must be shown that the pollutant has a direct influence on the useful life of the material. This concept is only recently gaining general acceptance in materials damage estimates.

B.2.4.5 Exposure--A critical element in determining dollar losses due to pollutant attack is the amount of material actually exposed to pollutants. In the past, that factor has been calculated by determining the amount produced during a period equal to the standard useful life of that material, followed by the application of an "exposure factor." The most serious flaw in this methodology is the assumption that once a material is in place it remains unchanged, except for deterioration. In some cases such an assumption may be warranted, but in others it totally ignores the effects of maintenance operations.

Galvanized steel is a good example. Not only has pollutant damage to galvanized steel been well studied, but such damage is included in many materials damage estimates. For many years galvanized surfaces were not coated because of poor paint adhesion. During the last 10 years, however, numerous paints and coatings have been introduced that not only adhere to galvanized steels but also offer excellent pollutant resistance (J. E. Kubanic, personal communication, 1978). Where bare galvanized steels are susceptible to pollutant attack, the coating of such materials (especially for roofing and siding applications) has become common. Therefore, although galvanized products are still in extensive use, the amount of zinc actually exposed to pollutants has greatly decreased--a factor not considered in most materials damage estimates to date.

It is important to note that air pollutant damage is basically a surface phenomenon. Once a material has been coated, the damage function to be considered is no longer that of the original material, but that of the coating. Figure B-5 illustrates a hypothetical case where a material of one susceptibility is coated with a material of different susceptibility to pollutant attack. The rate of damage of the original material is changed to the rate of damage of the coating. When the coating has deteriorated to the point that it no longer provides protection, the rate of damage may revert to or even exceed the original surface rate. However, it is common practice to repaint either before deterioration reaches ~10 percent or well before serious damage to the underlying material occurs (Steel Structures Painting Council 1977).

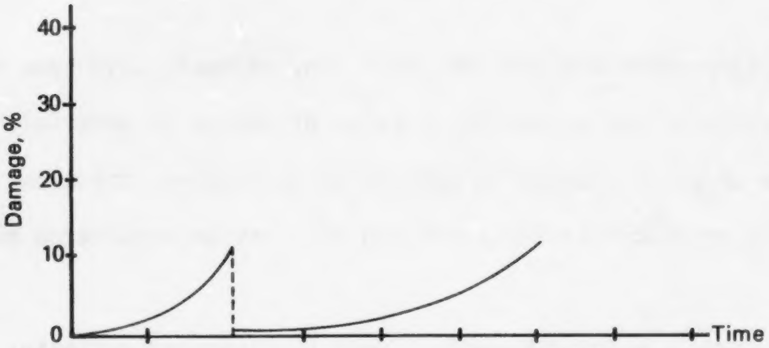

Figure B-5. Hypothetical rate of damage: original material vs. coating.

B.2.4.6 Social factors--The situation is further complicated for both the comparative and analytic approaches by the social component of maintenance schedules. For instance, although a house may need repainting, the required work might not be performed if the owner cannot afford it. On the other hand, a house not needing painting might be repainted because the owner wishes to change the color. It would be erroneous to compute a cost for maintenance that was warranted but not performed. Likewise, it would be wrong to ascribe a maintenance cost to the effects of air pollution when the reason for the cost-generating maintenance was something else.

B.2.4.7 Indirect costs--The indirect costs of materials damage have not been documented but may be hypothesized. For example, reduction in property values in areas that are perceived as polluted can be a major cost item, and obvious materials damage can play a major role in that perception.

B.2.5 Major Economic Studies

B.2.5.1 Introduction--Over the past 60 years many attempts have been made to estimate the costs of air pollution in terms of damage to materials and soiling. Because of basic changes in estimation techniques, these earlier studies are mainly of historical interest and will not be considered here.

B.2.5.2 Recent economic estimates--Recent work has improved estimates of materials damage costs, but there are still major deficiencies. Some of the most serious problems are deeply rooted in the fundamental strategy of the approaches used.

The most comprehensive recent review of the economic effect of air pollutants was assembled by Waddell (1974). The portion of this review devoted to the damage to materials was based on several studies using a number of approaches and covering various parts of the problem area. These studies are discussed briefly in the following sections.

B.2.5.3 Salmon: General materials--In terms of the number of materials covered, the study by Salmon (1970) is the most comprehensive attempted thus far. His study covered 32 materials and included a literature survey and interviews with selected persons. Costs were based on damaged properties of the materials or reduced serviceability. The economic value of a material exposed to air pollution was assumed to be the product of annual production in dollars multiplied by an average product life. To this value was applied an estimate of the net amount of deterioration occurring as a result of a fixed amount of air pollution in comparison with deterioration in a nonpolluted area. The results, in terms of dollar cost for each of 32 materials, were ranked according to total annual economic loss. The major purpose of this ranking was to show relative, rather than absolute, values for air pollution damage. Salmon listed the pollutants in order of decreasing economic importance, along with the materials they damage:

1. Sulfur oxides: metals, cotton, finishes, coatings, building stone, paints, paper, and leather
2. Ozone: rubber, dyes, and paints
3. Nitrogen oxides: dyes and paints
4. Carbon dioxide: building stone
5. Particulates: stone, clay, and glass

Salmon estimated costs of soiling to be $100 billion a year, a figure based on the total area of the material exposed, a unit cost for cleaning or repainting a surface, and the increased cost of cleaning or painting to maintain a surface in a "clean" condition. The standard used for "clean" was assumed to be that found in a "normal" suburban residential area. On the basis of the methods used and the assumptions made, Salmon's value for total soiling costs is not considered valid. Specifically, there is considerable overlap between soiling and other forms of damage; moreover, there are serious questions regarding the validity of presumed costs based on some arbitrary standard of cleanliness and willingness to pay for such upkeep.

To illustrate some of the problems in trying to use this estimate of soiling cost, Salmon showed that over one-third of the total soiling cost, or $35 billion, is for paint damage. The figure is questionable, since the total value at the manufacturer's level of shipments of paints, varnishes, and lacquers in 1968 was only $2.6 billion, or less than one-tenth of Salmon's value for soiling damage to paint.

B.2.5.4 Robbins: Electrical contacts--Robbins carried out a study in 1970 to determine whether air pollution causes economically important damage to electrical contacts. The categories of equipment covered included switches, relays, connectors, potentiometers, and commutators. The information for the study was collected through literature searches and discussions with manufacturers. The major cost categories covered were the direct cost of plating contacts with precious metals and the indirect costs associated with using air-cleaning equipment to provide switch gear with a pollution-free atmosphere. These costs are summarized as follows:

Plating contacts	$20 million
Air cleaning	25 million
Washing insulators	4 million
Research and development by firms affected by such damage	5 million
Costs of equipment failures	10 million
Total	$64 million

The author concluded that economic damage to electrical contacts was not as serious as had been thought. In any case, the principal finding of the work was that SO_2 and H_2S damage to low-voltage electrical contacts was more costly than all other types of damage to electrical contacts combined. However, it is not clear if these costs were incurred for prevention of pollution damage or for some other reason.

B.2.5.5 Fink: Corrosion of metals--In 1971, Fink and his associates assessed the costs of air pollution-induced corrosion to metallic systems and structures exposed to the outdoor atmosphere. The study was based on national shipment/value data for selected materials and articles and assumed that 80 percent of these materials were in "polluted" areas. Nine categories of materials were identified as being both economically significant and sensitive to air pollution damage. The total cost was made up of two components: the extra amount of protection and maintenance needed in "polluted" areas, and the cost due to shortened life from air pollution-induced corrosion.

The results of the study are summarized in Table B-13. Note that the first four items are principally galvanized structures and systems and that they account for about 93 percent of the total cost. The incremental costs of color-coated galvanized steel are not included in the estimate. The principal criticisms of this study are that damage is not related to specific levels of air pollutants; no attempt was made to account for the effects of relative humidity and its variations from one part of the country to another; also, maintenance factors do not include substitution of a paint surface for a bare galvanized one.

B.2.5.6 ITT: Electrical components--In 1971, the ITT Electro-Physics Laboratories studied the effects of air pollutants on electrical components. The study was similar in approach to that of Robbins and evaluated damage costs for semiconductors, integrated circuits, television picture tubes, connectors, transformers, relays, receiving tubes, and crystals. Results of this study indicated that particulate matter is more damaging to electrical components than is SO_2.

Table B-13. ANNUAL COST OF CORROSION BY AIR POLLUTION DAMAGE OF EXTERNAL METAL STRUCTURES, 1970

Structural system	Useful life years	Cost basis	Cost, \$ million	Percent of total
Outdoor metal work	45	Maintenance	914.0	63.0
Chain link fencing	30/20	Maintenance	166.0	11.5
Pole line hardware	30	Replacement	161.0	11.1
Galvanized wire and rope	20	Replacement	112.0	7.7
Steel storage tanks	50/11	Maintenance	46.3	3.2
Bridges	30	Maintenance	30.4	2.1
Street light fixtures	20	Maintenance	11.9	0.8
Power transformers	30	Maintenance	7.5	0.5
Transmission towers	30	Maintenance	1.5	0.1
Total			1450.6	100.0

Source: Fink et al. (1971).

Total damage costs attributed to air pollution (including sulfur compounds) were estimated to be \$15.5 million, of which \$2.3 million was for damage prevention (e.g., protective atmospheres) and \$13.2 million was for maintenance (e.g., cleaning, repairing, and replacement).

B.2.5.7 Spence and Haynie: Paints--In 1972, Spence and Haynie investigated the deterioration of exterior paints by particulate matter and by the combined effects of SO_2 and particulate matter. The authors developed costs for air pollution-induced paint damage principally on the basis of differences in service life between urban and rural areas. Of the \$704 million estimated total damage costs, \$540 million or over 75 percent was for household paint. "Normal service" life was based on Internal Revenue Service estimates. Such lifetimes, it should be noted, are often based more on depreciation schedules than on actual lifetimes.

B.2.5.8 Gillette: SO_x effects on general materials--In 1975, Gillette presented the results of a study based on the same general approach used by Fink et al., except that he considered a broader range of materials and limited his investigation to the effects of sulfur oxides. Further, Gillette used air quality data and concentration-response data for SO_2, considered the joint effects of humidity and SO_2 at various locations in the country, assumed that materials were distributed according to population, and considered the differences between indoor and outdoor exposures.

Table B-14 tracks Gillette's estimated SO_2 damage costs for the years 1968-72. Note that estimated damage costs dropped from \$909 million in 1968 to \$75 million in 1972. The author further estimated that SO_2 damage costs were probably greater than \$1 billion prior to 1968. He also showed that between 1968 and 1972 the estimated percentage of materials exposed to levels exceeding the primary annual average standard (80 $\mu g/m^3$) dropped from 25 percent to 3 percent.

B.2.5.9 National estimate of materials damage costs--Waddell summarized several studies to estimate nationwide costs of air pollution damage in 1970. The studies he considered are listed in Table B-15.

Table B-14. ESTIMATED MATERIALS DAMAGE COSTS ATTRIBUTED TO AMBIENT SO_2 LEVELS BY REGIONS OF THE UNITED STATES, 1968-72

	Damage costs, $ x 10^6				
Year	Northeast	North Central	South	West	Nationwide
1968	649.8	212.5	23.8	23.1	909.2
1969	430.3	193.2	12.2	13.1	648.8
1970	278.4	102.3	7.3	8.6	396.6
1971	46.7	42.1	8.8	10.5	108.1
1972	33.9	29.8	6.3	5.0	75.0

Note: Materials damage and SO_2 levels were calculated by Standard Metropolitan Statistical Areas (SMSAs). In each SMSA, the air quality measured at the center city site was assumed to be representative of the air quality for the entire SMSA.

Source: Gillette (1975).

Table B-15. ESTIMATES OF TOTAL COSTS FROM AIR POLLUTION DAMAGE TO MATERIALS IN 1970

Source of information	Material category	Principal pollutants	Estimated cost, $ billion
Mueller-Stickney	Elastomers	Ozone	0.5
Gillette	General materials but principally metal corrosion	SO_2	0.4
Salvin	Textiles & dyes	NO_2, O_3	0.2
Spence-Haynie	Paints	Particulates, SO_2	0.7
Salmon	Remainder of important materials not accounted for above	Various, but probably mostly SO_2	0.4
Total			2.2

Source: Waddell (1974).

Waddell assumed that the Gillette study is more defensible than the Fink study, that it does not significantly overlap the Spence-Haynie study, and that it includes damage to electrical contacts and components. If the materials evaluated in these studies (zinc, paints, rubber, carbon and alloy steel, fibers, cement and concrete, plastics, building brick, paper, leather, wood, and building stone) plus those believed by Fink to be relatively unaffected by air pollution (aluminum, copper, stainless steel, and lead) are subtracted from the Salmon study, a total remainder from the Salmon study of $0.4 billion is obtained. Using these data and the assumptions outlined above, Waddell estimated 1970 air pollution damage costs to be $2.2 billion.

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B.3 VISIBILITY AND CLIMATE

High atmospheric concentrations of sulfates reduce visibility, and the possibility that they play a role in climatic effects has also been considered. Mobile sources produce sulfates directly as well as indirectly, in that the sulfur dioxide they emit can be oxidized to sulfates in the atmosphere. However, these sources account for only a small portion of the total atmospheric sulfates, so small in fact that they would not in themselves measurably reduce visibility or exert any effects on climate. The following report defines relationships between sulfur-bearing compounds and visibility reduction and also addresses the question of climatic effects. Visibility reduction will be explored in greater detail in a separate, later report required under Section 169A of the 1977 Amendments to the Clean Air Act.

B.3.1 Visibility

Visibility in daylight is the greatest distance at which the unaided eye can see a prominent dark object against the sky at the horizon. This distance is strongly affected by the number of very small particles in the atmosphere. These particles, which cannot be seen by the unaided eye, produce light-scattering effects, thereby reducing visibility. Paradoxically, those particles which are large enough to be seen, such as dust from a quarry or an unpaved road, do not usually reduce visibility on a regional basis. To examine the relationship between mobile emissions of sulfur-bearing compounds and visibility, the following discussion will consider ways to measure visibility, various factors that lower it, and how sulfur-bearing compounds affect it.

The most common source of visibility data are observations taken at weather stations. When visibility is reported at weather stations, it is a composite value, called "prevailing visibility," for the greatest distance visible around at least 180 degrees of the horizon, not just for a single direction. For example, if the horizon were divided into four sectors, with visibilities of 3, 4, 5, and 8 miles, the prevailing visibility, or just visibility, would be recorded as 5 miles. Particularly low or variable visibilities in a given sector or direction would be noted as additional data. Reporting of prevailing visibility has been a standard procedure in the weather service since 1939.

This reporting procedure, however, has certain limitations. For instance, since most visibility observations are taken at airports, usually located outside of the downtown and industrial areas, the prevailing visibility observation may not accurately describe the visibility in more polluted urban areas. Also, many weather stations do not have suitable markers beyond 15 miles; greater visibilities are reported as 15+. For locations characterized by very clear, or clean, atmospheres such as in remote areas, there frequently is a need for visibility markers at great distances, since objects can often be discerned at a distance of 100 miles. In flat country, observation distances are inherently limited by the curvature of the earth. For instance, at sea the horizon for an observer who is 6 ft above the water is 5.2 km (slightly more than 3 miles). Where there are no suitable distant visibility targets, long-range visibilities are usually estimated from the clarity of objects, requiring very subjective evaluations. Therefore, the large and valuable body of meteorological observations must be utilized with care if the data are to establish relationships between air pollution and visibility.

Visibility is reduced by light scattering, which impedes the transmission of light and weakens contrast. The reduction of light in the atmosphere by light scattering and by light absorption is termed "extinction." Light scattering is caused primarily by particles or aerosols, but even in their absence, visibility would be limited by the scattering of light by air molecules (so-called Rayleigh scattering). (Note: An aerosol is a gas-phase colloid consisting of small suspended particles uniformly dispersed in a gas.)

The particles that scatter light with greater effectiveness range primarily between 0.1 and 1.0 μm in diameter. (A micrometer, or micron, is one-millionth of a meter, or a thousandth of a millimeter.) Particles forming aerosols in the atmosphere can be divided into three groups according to their method of formation. The smallest particles, having a diameter of about 0.04 μm or less, are produced mainly by gas-to-aerosol conversion (e.g., sulfur dioxide to sulfates) or from the condensation of vaporized materials (e.g., oils or metal vapors). This size range is termed the "nuclei" mode. At the other extreme are the "coarse" aerosols with particles larger than 2 μm, which result from abrasive processes (e.g., grinding and atomization) and include almost all of the natural particulate matter in

Table B-16. ATMOSPHERIC AEROSOLS: NAMES AND CHARACTERISTICS[a]

Name	Dispersed phase	Primary origin	Size (μm)[b]
Smoke	Solid or liquid	Condensation[c]	<1
Mist	Liquid (water droplets)	Condensation or dispersion[d]	5-1000
Fog[e]	Liquid (water droplets)	Condensation	2-30
Haze	Solid-hygroscopic (exists in lower RH than fog)	Condensation nuclei	<1
Dust	Solid	Dispersion	>1
Aitken (condensation nuclei)	Solid or liquid	Combustion, atmospheric chemistry	<0.1
Ice nuclei	Special crystals	Natural dusts	>1
Atmospheric ions	Electrically charged particles	All sources	0.001-.01
Coarse particles		Dispersion	>2
Fine particles[f]		Condensation, coagulation	<2

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[a] Other nomenclature for describing atmospheric conditions: smog = smoke + fog; smaze = smoke + haze.
[b] Visibility is reduced primarily by particles measuring 0.1 to 1 μm in diameter; the average wavelength of visible light is 0.55 μm.
[c] Condensation is the physical process by which a vapor becomes a liquid; opposite of evaporation.
[d] Dispersion is the distribution of finely divided particles in a medium.
[e] Fogs are clouds that make contact with the earth's surface.
[f] Divided further into ultrafine particles (<0.1 μm).

wind-blown dust and sea spray (Whitby 1978). The third size range is the "accumulation" mode, which includes particles from about 0.1 to 0.5 μm. These particles result primarily from the coagulation of the smaller-sized aerosols. Such aerosols generally do not settle out by gravity and are not removed efficiently from the atmosphere except by incorporation into clouds and the subsequent rainout. Studies show that they may persist in the atmosphere for several days. Of the three size classes, the accumulation mode is mainly responsible for the reduction of visibility and the alteration of solar/terrestial radiation. Table B-16 lists the major atmospheric aerosols of both natural and anthropogenic origin.

Another cause of visibility reduction is the interaction of aerosol particles (either natural or anthropogenic) with atmospheric water vapor. It has been shown that a relative humidity (RH) range from 30 percent to about 60 percent frequently has little or no effect on light scattering (and, hence, visibility). However, the light-scattering effects double by the time the humidity reaches slightly above 80 percent. The effect is even more dramatic at near 88 percent RH, where light scattering increases to four times the level measured in the dry air (30 percent RH). This effect arises in part from hygroscopicity of particulate materials. As the humidity increases, sulfate and other particles pick up water, thereby increasing in size. As they reach a size corresponding to the wavelength of visible light and increase further to become fog droplets, their potential to scatter light and thereby reduce visibility is maximized. It may be useful to note that the effect of RH may vary considerably depending on the composition of the aerosol.

Trijonis and Yuan (1978) noted the visibility effects of changes in sulfur emissions from copper smelting. During a 9-month strike in the copper-smelting industry in Arizona in 1967-68, sulfate levels in the air were 38 to 76 percent lower than seasonal averages at five sites within 70 miles of copper smelters. Grand Canyon and Mesa Verde National Parks, about 200 to 300 miles from the smelters, also experienced significant drops in ambient sulfate levels. During the strike, visibility reportedly improved from 5 to 25 percent at locations within 50 miles of the operations. Pollution potential from other sources appears to have been slightly greater than usual during the 9-month period. Since weather patterns were about normal, Trijonis and Yuan concluded that weather was not a major factor contributing to the improved visibility.

Visibility can be measured by mechanical means. The most widely deployed instrument in the meteorological services is the transmissometer, which measures the transmitted light along a path of known length (usually 500 ft). It utilizes an intense beam of carefully focused, pulsating light which is measured by a photocell receiver. At selected light pulsation frequencies, this instrument works satisfactorily both day and night. In usual practice, however, the instrument is designed and installed so as to sense very low visibilities, such as are due to fog at airports. As such, the instrument may not be accurate enough for the higher light transmission values necessary in air pollution evaluations.

Telephotometers can be used to measure contrast differences over distances much greater than those a transmissometer can measure directly.

The contrast of objects on the horizon can be measured. Whereas the measurement of a transmissometer is limited to the fixed path between the light source and receptor (or reflector), the measurement of a telephotometer may be made in any direction from the point of observation.

Photographic techniques to determine visibility measure reductions in contrast caused by haze. They can operate over a shorter light path than the transmissometers but usually are directed toward more distant visibility targets (Steffens 1949).

The integrating nephelometer (Charlson et al. 1969) is increasingly deployed for visibility measurements. This instrument draws air into a sample chamber, where it is illuminated by a pulsed flash lamp, and the scattering of light is detected by a photomultiplier tube. It is used both in research and as a routine monitor of the atmosphere's scattering potential.

Since the magnitude of scattering from a particle is a function of the wavelength of the incident light, it is possible to use scattering intensities at two or more wavelengths to determine the average size distribution of an aerosol. Because the nephelometer samples only a small volume of the atmosphere and senses only scattering effects, it can give misleading results if it is not properly calibrated and if the concentrations in the air are not relatively uniform. Nevertheless, it is the best indicator of visibility currently available for routine use in many air pollution monitoring situations.

More recently, laser transmissometer systems have been developed to measure visibility over long distances. Although these systems require more refinement, the coherent nature of the light intrinsic to the laser can permit quantitative detection of aerosol attenuation over longer ranges. Also, the use of laser light of different frequencies can assist in determining what particle sizes are contributing to visibility reduction.

Because of the importance of aerosols in visibility reduction and other consequences of air pollution, EPA has supported a great deal of research in this area. EPA is currently testing and evaluating alternative means of visibility measurement, and there is considerable debate over which is the best visibility indicator. Another line of research has resulted in a "dichotomous" mass sampler, which collects ambient air samples in two size fractions. One is termed the "fine fraction," containing particles less than 3.5 µm. The other, the "coarse fraction," contains particles ranging from 3.5 to 15 µm in diameter. This instrument has been extensively used in recent aerosol research studies (Stevens et al. 1978).

The studies of the size distribution of aerosols have been further extended by the collection of enough material in the various size ranges to permit chemical analysis by mass spectrometry, X-ray fluorescence, laser-induced Raman spectroscopy, and other advanced techniques. These complicated procedures require careful material collection and handling methods. A body of data is just now becoming available with sufficient geographical, seasonal, and source information to permit some generalizations to be made.

First, most of the sulfur occurs in the "fine" particulate matter (i.e., the "accumulation" and "nuclei" modes). Further, in some areas, sulfur as sulfate can account for much of the mass of this fine material. Depending upon the situation, the bulk of the remaining mass of material in this optically active range may be nitrate (NO_3) or organics. A complete description of the specific molecular forms containing sulfate and nitrate ions is not yet available. However, discussions of the various species of sulfur have been described by Charlson et al. (1978). Knowledge of the specific sulfur compounds and of their distribution and conversion rates would enable much more precise determination of their effect on visibility. Compounds in the atmosphere known to reduce visibility are ammonium sulfate, sulfuric acid mist, and ammonium acid sulfate.

A Los Angeles study relating aerosol composition to light-scattering measurements (White and Roberts 1977) indicated that sulfates and nitrates always dominate the scattering but that one may be more important than the other, depending on the location of measurement. Whether these relationships hold in other geographic areas, at different pollution concentrations, with different aerosol compositions, and with different meteorological conditions remains to be seen. Extrapolations from a study site to another geographic area should be done with caution. However, most available studies indicate that sulfates should be considered a primary cause of visibility reduction (Trijonis and Yuan 1978; Leaderer 1978). The added contribution from nitrates seems to vary according to relative humidity.

Data taken in controlled laboratory experiments can systematically be used to investigate the physical/chemical mechanisms that lead to results observed in the ambient atmosphere. EPA sponsored a study of such mechanisms, entitled "A Methodology for Determining the Effects of Fuels and Additives on Atmospheric Visibility" (Kochmond et al. 1975). Automobile exhaust with incremental hydrocarbon and diluted with ambient air was introduced into a large smog chamber and irradiated with simulated sunlight. Visibility, expressed in terms of nephelometer light-scattering values, was measured after various times of irradiation. Variables in addition to irradiation time were humidity, amount of sulfur dioxide, and the amount of particulate matter. Early in the test period, it was observed that the ambient air introduced to dilute exhaust emissions exerted marked and variable influences on visibility measurements. This effect was due to shifts in the background concentration of visibility-reducing aerosols. Filtered air was therefore substituted to enable strict control of the experimental values.

The results of these tests demonstrated the contribution of a number of factors to the visibility-reducing effects of automobile exhaust. While the ratio of HC/NO emissions was important, it was shown that visibility reduction was also correlated with SO_2 and relative humidity (RH). Under the controlled conditions of the test, the effects of these two factors, which were interactive, increased with irradiation time. As shown in Figure B-6, when RH was low (35 percent), the exhaust emission's effect on visibility changed little over the course of the irradiation period. At the other extreme, when the RH was raised to 80 percent and the air-exhaust mixture was supplemented with additional SO_2 (0.9 ppm), visibility fell from an initial value of about 50 km to less than 4 km within 22 hr.

Analysis of data from tests of different fuels indicated that the sulfur content of gasoline was directly related to emissions which led to the formation of light-scattering aerosols. Thus, at a given ratio of HC/NO emissions, fuels with high sulfur content had the most marked effects on visibility in this chamber study.

Although laboratory data provide important information on specific mechanisms of aerosol formation and its effect on light scattering, it is necessary to obtain information on the effects of ambient aerosol formation on visibility. Many visibility studies have centered on the Southwestern United States. However, these studies may not be applicable to other geographic locations. When considering light-scattering effects in the atmosphere, it should be noted that visibility may not be strongly related to the total mass of suspended particulate material. Those particles having characteristic dimensions near the wavelength of visible light are more effective in light scattering, and the proportion of these particles to the total particulate mass will vary with time and place. Therefore, arbitrary correlations between visibility and total suspended particle mass are not likely to be constant when different regions or seasons are compared. Relationships between visibility and particle mass have been shown to have an accuracy of plus or minus a factor of two.

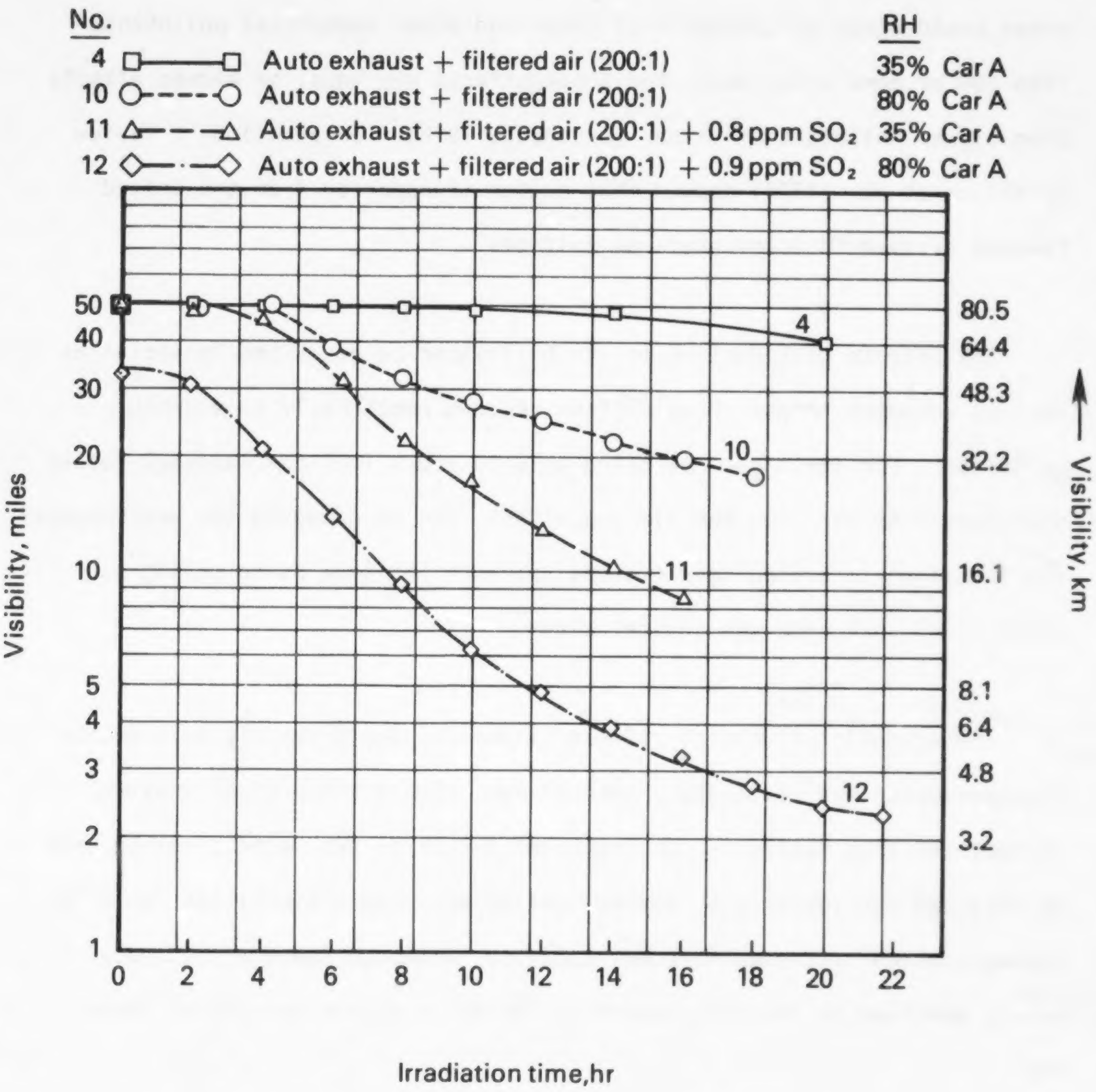

Figure B-6. Effect of relative humidity and added SO_2 on test results.

There is also concern with transport of sulfates into relatively unpolluted areas from long distances. Impairment in visibility in these areas could occur by transport of urban and other industrial pollutants from 200 or more miles away, and these effects may equal or exceed effects from closer pollutant emissions (Ursenbach 1978). In addition, a review by Wilson et al. (1977) showed that sulfur transported from the United Kingdom arrived in Scandinavia as sulfates.

The effects of reduction of visibility can be described in social as well as economic terms. People often respond emotionally to visible pollution. For instance, not being able to see a familiar landmark raises such questions as: "Is the air unhealthy? Are we damaging the environment?" The reactions to seeing smog effects are much the same as to seeing a dirty river that once was crystal clear.

The economic effects of reduced visibility are primarily felt in the transportation and real estate enterprises. The ability of an area to attract and keep residents, an important factor in real estate values, can be impaired considerably by obvious pollution. Low visibilities can slow transportation systems and reduce capacity, although these effects are mostly confined to the very lowest visibilities characteristic of heavy fog.

B.3.2 Climate

Pollutants released to the atmosphere alter the environment in ways other than visibility reduction. Importantly, they may lead to slow and subtle changes in atmosphere composition and, possibly, climate.

Climate has been defined as the long-term manifestations of weather at a given location over a specified period, usually several decades; current discussions of climate usually use the reference period 1940 to 1970. Weather manifestations may be specified as averages (i.e., of temperatures, rainfall, wind speeds, etc.) or as frequencies (i.e., hurricane occurrence, maximum temperature over 100°F [36°C], etc.).

Climate exerts a major influence on many aspects of society, including architecture and agriculture. Climate factors such as temperature and precipitation must be considered both in design of buildings and in crop selection. Some acts of man may increase the impact of climatic trends. For example, the recent drought in Northwest Africa may have been exacerbated by overgrazing the land during periods of above-average rainfall.

Societal responses to long-term climate changes have not been explored fully, but it is obvious that any major shift in food production capability of a large area would be socially disruptive, as was proven by the Dust Bowl of the 1930's. Recently, much attention on an international scale has been devoted to both the effects of long-term climate changes on society and, conversely, society's influence on long-term climate changes.

These issues were addressed at the 1972 United Nations Conference on the Human Environment. The second of these issues is of present concern--the effects of air pollution on climate.

Climate change attributable to air pollution may be effected in a number of ways. The release of gases and production of aerosols could significantly alter the atmosphere's composition (hence, heat-absorption characteristics) to bring about a change in the earth's radiation balance. Shifts in this balance would be associated with temperature change. Cloud formation could also be affected. Changes in cloud cover could alter albedo (reflection of sunlight), another important factor in maintaining the earth's radiation balance. Changes in the location, frequency, and amount of precipitation might also occur.

So far as is known, only sulfur compounds in solid phase (i.e., sulfates) are involved in processes affecting climate. In fact, for all practical purposes, this discussion can be limited to sulfate particles. These particles can absorb sunlight and water from the atmosphere, and they can also affect cloud condensation nuclei (CCN), which are critical in the rainfall process. Many reports have correlated particulate matter with potential climate change. Natural events such as the explosive injection of volcanic material into the atmosphere verify this correlation--Krakatoa in 1883, Katmai in Alaska in 1912, and, more recently, Bali's Mt. Agung in 1963. These eruptions showed the capacity of volcanic material to scatter incoming solar radiation. At the time of Agung, monitoring systems had progressed to the point that the spread-and-time history of the aerosol could be documented with the aid of laser systems. An analysis of upper-air temperatures in the Southern Hemisphere detected a major warming attributed to the absorption of radiation by the aerosol. Little surface temperature or rainfall effects have been identified directly with these events. In general, however, the large variability of weather obscures all but the most dramatic effects.

On a global scale, meteorological observations linking air pollution to climate changes are lacking. Over cities, the reduction of received radiation can be ascribed definitely to the increase of fine aerosols. Cloudiness appears to be increased by 5 to 10 percent. Fog develops about twice as often over urban areas as over the countryside, and the duration is lengthened by an abundance of hygroscopic compounds. Thunderstorms may be enhanced, and precipitation appears to be increased over and especially downwind of cities. The effects of pollutants on precipitation have yet to be separated from the effects of thermal energy over urban areas. In studies of the correlates of air pollution and local or regional climatic processes, a role for mobile source emissions has not been defined.

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